EFFECT OF SOURCE OF TRACE MINERALS AND PLANE OF NUTRITION ON GROWTH AND HEALTH PERFORMANCE AND HOOF DEVELOPMENT OF TRANSPORTED DAIRY CALVES

BY

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THESIS

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ABSTRACT

The aims of the present experiment were: 1) to evaluate the effects of enhanced feeding programs and trace minerals source early in life on growth and health of transported calves, and 2) to quantify the effect of early nutrition programs and trace minerals source on hoof tissue development in young transported calves.

Ninety Holstein bull calves <1 wk old were assigned to treatments in a 2 x 2 factorial arrangement of plane of nutrition (PN) and trace minerals source (TM) in a randomized, complete-block design. Conventional PN received milk replacer (22% CP, 20% fat; 568 g/d powder for wk 1 to 4, 284 g/d powder for wk 5) plus ad libitum starter mix (18% CP, DM basis, for wk 1 to 12) and hay (0.5 kg/d as fed, for wk 10 to 12) and were weaned at 6 wk. During wk 13 to 20 calves were fed 3.2 kg/d of grower mix (16% CP, DM basis) plus chopped hay ad libitum. Accelerated PN received variable amounts of milk replacer (28% CP, 20% fat; 810, 1136, and 568 g/d of powder for wk 1, 2 to 6, and 7 respectively) plus ad libitum starter mix (22% CP, DM basis wk 1 to 12) and limited hay (0.5 kg/d as fed, for wk 10 to 12) and were weaned at wk 7. Ad libitum grower plus limited chopped hay (0.5 kg/g, as-fed basis) was offered from wk 13 to 20. Milk replacer was formulated to contain balanced amounts of either inorganic or organic TM (50, 50, 10, and 100 ppm of Zn, Mn, Cu, and Fe, respectively).

Accelerated PN treatments increased overall growth, average daily gain (ADG), and gain to feed (G:F) through wk 20. Organic TM increased growth when supplemented to accelerated PN but not when supplemented to the conventional programs. Therefore, a synergistic effect was observed between accelerated PN and organic TM by means of increasing the biological value of each other.

Calves are born with uneven claw lengths; rear medial claws were longer than front and lateral claws. Organic TM increased claw length after wk 10 in comparison to inorganic TM, and in the same way, organic TM increased groove length when fed in enhanced nutritional programs but not in conventional. These effects might be associated with an accumulative effect of organic TM supplementation. Enhanced PN programs had a greater effect in increasing overall hoof growth; in contrast, organic TM had a greater effect on reducing overall hoof wear.

Results indicated that enhanced nutritional programs during early life will allow calves to have greater overall growth and maintain a normal health status throughout the preweaning period. Accelerated nutritional programs promoted a greater effect of organic TM on hoof growth, while organic TM to decreased overall hoof wear compared with inorganic sources.

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A	accelerated
AO	accelerated organic
ADF	acid detergent fiber
ATP	adenosine triphosphate
ADP	apparent digestible protein
AOAC	Association of Official Analytical Chemists International
ADG	average daily gain
BW	body weight
cm	centimeter(s)
CL	claw length
C	conventional
Cu	copper
CP	crude protein
Cys	cysteine
d	day(s)
dL	deciliter(s)
°C	degrees Celsius
DCAD	dietary cation-anion difference
DM	dry matter
DMI	dry matter intake
G:F	gain to feed ratio
g	gram(s)

GL	groove length
His	histidine
h	hour(s)
IgG	immunoglobulin
I	inorganic
ICS	intercellular cementing substance
Fe	iron
kg	kilogram(s)
L	liter(s)
Mn	manganese
Mcal	megacalorie(s)
ME	metabolizable energy
Met	methionine
mg	milligram(s)
mm	millimeter(s)
NAHMS	National Animal Health Monitoring System
NRC	National Research Council
NE_g	net energy of gain
NE_m	net energy of maintenance
NDF	neutral detergent fiber
N	nitrogen
O	organic
ppm	parts per million
%	percent
PN	plane of nutrition
P	probability

RNA	ribonucleic acid
n	sample size
TDN	total digestible nutrients
TM	trace minerals
wk	week(s)
Zn	zinc

INTRODUCTION

The newborn calf presents challenges for the dairy producer. The producer must strive to minimize costs and maximize profit by increasing growth performance, reducing feeding labor, decreasing mortality, decreasing days to first calving, decreasing health treatment costs, and increasing productivity once the heifer enters the milking herd. In contrast, the newborn calf needs to adapt to a completely new environment loaded with pathogens, must thermoregulate its own body, must change its digestive system from umbilical nutrients delivery during gestation to digesting bottle- or bucket-fed milk, and then must transition from pre-ruminant to ruminant. After weaning, the animal will not face such a stressful period again until it reaches parturition and enters the milk herd.

According to Wolf (2003), as dairy farms become larger, the use of off-site calf raising operations is more common. This observation is supported by the increase from 7.2% to 11.5% in heifers sent off-site to be raised regardless of dairy farm size, as reported by the USDA National Animal Health Monitoring System (NAHMS) during 2002 (USDA, 2002) and 2007 (USDA, 2007). Also, USDA-NAHMS during 2007 reported that 46% of large dairy farms (>500 cows) raised their heifers off-site (USDA, 2007). Therefore, decreasing negative effects of transportation stress, bad colostrum management, and environmental pathogens is important to decrease mortality rates and improve growth performance.

Adequate management of early nutrition, environment, and health of the calf has been proposed to ameliorate stress factors after birth (Davis and Drackley, 1998). Early nutrition in dairy calves has become a polemic subject with arguments for both minimizing milk replacer and feeding larger amounts of milk replacer. Minimizing cost by feeding small amounts of milk

replacer or whole milk has been encouraged because it increases dry feed consumption earlier in life, and may be more desirable for early weaning. On the other hand, enhanced early nutrition is based on providing increased amounts of milk replacer of higher protein content, which will boost early growth and reduce age at first calving so that producers might achieve greater economic benefits in the long-run. Enhanced nutritional programs promote greater rate of gain and efficiency of gain for calves fed milk (Huber et al., 1984; Jasper and Weary, 2002; Jensen, 2006; Khan et al., 2007a; Khan et al., 2007b; Borderas et al., 2009) or milk replacer (Diaz et al., 2001; Blome et al., 2003; Bartlett et al., 2006; Hill et al., 2006a; Hill et al., 2006b) at increased rates during the milk feeding period.

Trace minerals play critical roles in structural development (such as the hoof) and function of many cellular systems, one of which is the immune system. It is imperative, therefore, that adequate trace minerals status be established quickly after birth and maintained to ensure sufficient stores are available for optimal animal performance and so animals can withstand disease challenge or other stresses. "Bioavailable" or "organic" trace minerals have been demonstrated to enhance health status in dairy cattle, especially during times of stress such as the periparturient period (Ballantine et al., 2002; Nocek et al., 2006; Griffiths et al., 2007; Siciliano-Jones et al., 2008). Because of the greater nutrient demands for growth, biological and economic benefits of organic trace minerals might be more pronounced in accelerated early nutrition programs for calves.

First calving is the next extreme challenge period after weaning for the replacement heifer. During this time the animal will face a series of hormonal, physiological, and behavioral changes that without proper management and nutritional knowledge can lead to impairment of health status. According to NAHMS, lameness was the second most common health problem

and the first cause of death in dairy cows during 2006 (USDA, 2007). Lameness in dairy cattle has been well described in several studies as the result of different conditions that lead to a common locomotion problem and, consequently, is a major direct cause of economic losses in the dairy industry (Hendry et al., 1997; Green et al., 2002; Donovan et al., 2004; Neveux et al., 2006). Research has observed that cows identified as lame for the first time have a predisposition to recurrence of lameness or claw disorders in subsequent lactations (Peterse, 1986; Enevoldsen et al., 1991). Thus, anything that could be done during the rearing phase to prevent cows from becoming lame after first parturition must be a key management objective. Currently, data are limited on the effects of bioavailable trace minerals supplementation to dairy calves during the rearing phase on claw growth and health as well as any effects during the subsequent lactation.

The objectives of this experiment were: 1) to determine the effects of plane of nutrition and trace minerals source on growth and health in transported dairy calves, and 2) to evaluate the effects of plane of nutrition and trace minerals source on hoof health and development in dairy calves.

CHAPTER I

LITERATURE REVIEW

NUTRIENT REQUIREMENTS

Energy

Energy-yielding nutrients need to be provided to fuel maintenance functions and to allow growth in calves. Differences in energy requirements have been established depending on the phase of digestive function development: young preruminant calf fed only milk or milk replacer, calves fed milk or milk replacer and starter feed, and ruminant calves (NRC, 2001). The National Research Council (NRC) system (2001) uses units of metabolizable energy (ME) to define energy requirements.

Energy requirements are usually partitioned into ME of maintenance and ME of growth (Davis and Drackley, 1998). Fasting heat production is considered the minimum level of metabolism or heat production by the animal and is used as the starting point in deriving the maintenance energy requirements in the animal (Brody, 1945). The ME of growth is the ME needed to furnish the net energy of growth (NE_g), which is a function of energy stored in body tissues, primarily as protein and fat. Because every unit of protein deposited in tissue is accompanied by about 3 units of water, the gain of live weight in preruminant calves is driven by gain in protein rather than fat (Roy, 1970). Increasing desired rates of gain increases the demands for ME, as does increased maintenance demands during cold stress (NRC, 2001; Nonnecke et al., 2009) or transportation stress (Ariel et al., 1995).

Energy requirements for maintenance and body weight gain for calves fed milk or milk replacer plus starter feed are the same for those of preruminant calves. Yet, the efficiency of utilization of ME is considered to be lower for transition calves (liquid plus dry feed) because they lose energy in heat of fermentation, plus must expend additional energy in prehension, mastication, and rumination, which increase the metabolic heat production (Davis and Drackley, 1998). According to the NRC (2001), estimated energy requirements for heifers from 100 to 500 kg of body weight were derived using the same methodology as described in preruminants and transition calves, although digestibility, metabolizability, and efficiency of ME use are all lower.

Protein

Protein requirements are partitioned into maintenance and gain (NRC, 2001). Maintenance requirements account for obligatory nitrogen losses in urine and feces, whereas gain is associated with nitrogen stored in tissues as protein. Protein requirements are expressed as apparent digestible protein (ADP), which is a function of endogenous losses plus nitrogen stored in liveweight gain divided by biological value of protein consumed (NRC, 2001).

The protein requirement for maintenance is estimated from metabolic fecal nitrogen losses and endogenous urine nitrogen losses. Metabolic fecal nitrogen is non-dietary nitrogen found as sloughed gut cells, bacterial debris, and digestive secretions. Endogenous urine nitrogen losses are products of tissue metabolism and turnover, and are expressed on the basis of tissue metabolism relative to metabolic body size ($kg^{0.75}$) (Davis and Drackley, 1998).

Total ADP required is the sum of requirements for maintenance plus gain but is mainly driven by the rate of body weight gain. Higher rates of body weight gain will require

increased amounts of ADP to synthesize protein in the tissues. The NRC (2001) used a constant 30 g N retained/kg of body weight gain or 188 g of protein accreted per kilogram of body weight gain based on previous literature (Blaxter and Wood, 1951; Brisson et al., 1957; Roy, 1970; Davis and Drackley, 1998). Conversely, Diaz et al. (2001) suggested that the 1989 Dairy NRC underestimated the retained protein at higher rates of body weight gains (NRC, 1989). Higher rates of body weight gain with improperly balanced diets will affect composition of gain and eventually influence the protein requirement of the calf. For example, Bartlett et al. (2006a) found that calves fed the same amount of ME but with protein limiting accreted more fat in body weight gain than calves fed adequate protein.

Trace minerals

Iron. Iron is a component of most known living organisms. In mammals, the main function of Fe is to hold together the hemoglobin molecule, which is responsible for transport of oxygen from lungs to all tissues of the body. Iron is involved in several other functions such as carbon dioxide removal from tissues, oxygen storage as myoglobin, and as a component of enzymes such as catalase and peroxidase (Beard, 2006).

The physical manifestation of Fe deficiency is presented as generic symptoms of anemia: tiredness, lassitude, and general feelings of lack of energy (Beard, 2006). Also, Heidarpour Bami et al. (2008) reported a decrease in red blood cells and weight gain when neonatal calves were not supplemented with Fe via injection.

During Fe overload the primary organs affected are the liver, heart, and pancreatic beta cells. All are tissues with highly active mitochondria, and impairment of mitochondrial functions and damage and rupture of lysosomes are the main cellular effects

during Fe toxicity (NRC, 2005). In the particular case of dairy calves, Jenkins and Hidiroglou (1987) did not register any negative effects when supplementing a milk replacer with Fe up to 5,000 mg/kg of DM, after which point an adverse effect on gain and efficiency of feed conversion was observed.

The NRC (2001) suggested a concentration of Fe in milk replacer and starter feed of 100 and 50 mg/kg of DM, respectively. Ceppi and Blum (1994) observed that feed intake, average daily gain, and growth:feed ratio were decreased in intact veal calves fed milk replacer containing 10 mg of Fe/kg of DM. Whole milk contains about 3 mg of Fe per kg of DM; thus, it does not provide enough Fe to sustain high growth performance and normal health status early in life.

Manganese. Manganese is considered an essential nutrient because it is known to function as an enzyme activator and to be a constituent of several metalloenzymes (Nielsen, 2006). Manganese-activated enzymes are involved in proteoglycan synthesis and hence bone formation. In addition Mn activates arginase and mitochondrial superoxide dismutase, both of which are manganese metalloenzymes (NRC, 2005).

Deficiency signs for Mn are variable depending on the animal species and include depressed growth and testicular degeneration in rats, slipped tendons in chicks, severe glucose intolerance in guinea pigs, and ataxia in mice (Nielsen, 2006). Similarly, Hansen et al. (2006) observed an adverse effect on fetal growth and development in pregnant heifers not supplemented with manganese when compared with heifers supplemented with 50 mg of Mn/kg of DM.

Manganese is considered to be one of the least toxic of the essential elements (NRC, 1980). Thus, most Mn toxicology studies are chronic dietary exposure studies rather than single dose experiments. Results from Cunningham et al. (1966) and Jenkins and Hidiroglou (1991) indicated that Mn above 1,000 mg/kg of DM concentration in milk replacer produced negative effects on growth, weight gain, and feed intake. Furthermore, 100% mortality was observed when calves were fed milk replacer supplemented with 5,000 mg of Mn per kg of DM (Jenkins and Hidiroglou, 1991).

The NRC (2001) suggested a concentration of manganese in milk replacer and starter feed of 40 and 40 mg/kg of DM, which concurs with recommendations of Davis and Drackley (1998) who recommended 10 to 50 mg of Mn/kg of DM.

Zinc. Zinc is well known for its essential characteristics as a nutrient for all animals, having a large effect on enzyme systems, protein synthesis, carbohydrate metabolism, and nucleic acid metabolism (Miller, 1970). Zinc also is a determinant of immune system development and maintenance of host defense from nitric oxide and oxygen radicals (Cousins, 2006).

The clinical signs of zinc deficiency include reduced growth, feed intake, and feed efficiency, as well as listlessness, reduced testicular growth, and parakeratotic lesions with greater effect on the legs, head, and around the nostrils (Ott et al., 1965). Also, swollen feet with open scaly lesions developed followed by alopecia and general dermatitis, which is consistent with a key role of zinc associated with the hoof keratinization processes in cattle (Tomlinson et al., 2004).

Acute toxicity of zinc through feed exposure has been associated with gastrointestinal distress with clinical signs of nausea, vomiting, abdominal cramps, and diarrhea (NRC, 2005). Chronic toxicity of zinc is difficult to relate to the physical and chemical properties of this element because it is not believed to be carcinogenic, mutagenic, or teratogenic, and it does not have a classical genetically caused storage disease (NRC, 2005). However, Jenkins and Hidiroglou (1991) reported negative effects on feed intakes, weight gains, and feed efficiency when feeding calves with a milk replacer supplemented with more than 700 mg of Zn/kg of DM.

Recommended concentrations of zinc range from 20 to 50 mg/kg of DM (Davis and Drackley, 1998). The NRC (2001) suggested an inclusion of 40 mg of Zn/kg of DM for both milk replacer and starter feed.

Copper. Copper was reported to be essential for growth and hemoglobin formation in rats in 1928 (Underwood and Suttle, 1999). Subsequent studies indicated that copper was an essential component of a number of enzymes such as cytochrome oxidase, lysyl oxidase, superoxide dismutase, tyrosinase, and ceruloplasmin (Linder and Massaro, 2002). Copper-dependent enzymes function in several processes including energy metabolism, maturation and stability of collagen and elastin, pigmentation, the antioxidant defense system, and iron metabolism (NRC, 2005).

Severe copper deficiency in mammals manifests in hypochromic anemia that is refractory to iron supplementation, neutropenia, thrombocytopenia, and hypopigmentation, plus anatomical and functional abnormalities in the skeletal, cardiovascular, and immune systems (Uauy et al., 1998). Copper deprivation during the fetal and neonatal periods also causes neurological abnormalities (Prohaska, 2006). Heidarpour Bami et al. (2008) observed a decreased weight

gain in calves that did not receive an injection of 160 mg of Cu at day 14; therefore, parenteral supply of copper during fetal development might not be adequate to sustain high performance in weight gain early in life.

Acute copper toxicosis signs include nausea, vomiting, diarrhea, excessive salivation, abdominal pain, convulsions, paralysis, and sometimes death (NRC, 1980). Necropsy performed in animals with clinical diagnosis of copper toxicosis reveals acute gastroenteritis, necrotic hepatitis, and splenic and renal congestion. According to the NRC (1980) the acute toxic level of oral copper is 200 mg/kg of BW in cattle. Previous literature reported adverse effects such as reduced intake and gain, apathy, anorexia, and brownish-red urine when feeding newborn calves for 6 wk with milk replacers containing up to 1,000 mg of Cu/kg of DM compared with calves fed 50 mg of Cu/kg of DM (Jenkins and Kramer, 1989).

Finally, when the calf transitions from pre-ruminant to ruminant it is important to notice the interaction of copper with other minerals such as sulfur and molybdenum. When concentrations of molybdenum and sulfur increase then copper bioavailability is reduced greatly, which increases the risk of copper deficiency. In contrast, lower concentrations of molybdenum and sulfur increase the risk of copper toxicosis (NRC, 2005).

The NRC (2001) proposed a concentration of Cu in milk replacer and starter feed of 10 mg/kg of DM for both feeds. Davis and Drackley (1998) reported a range from 5 to 20 mg of Cu/kg of DM from previous studies.

Accelerated versus conventional nutrition programs

Reduction of feeding cost for raising replacement heifers has been the main objective of the conventional feeding programs widely adopted by producers that want to reduce the length of the milk-feeding period. However, consistent improvements in growth, average daily gain (ADG), and feed efficiency of calves fed so-called "accelerated" nutritional programs have been reported throughout research addressing the problem during the past decade (Diaz et al., 2001; Blome et al., 2003; Bartlett et al., 2006; Hill et al., 2006a; Hill et al., 2006b). The extent of the effects of accelerated nutritional programs on health status early in life and economical impact on long-term performance is yet poorly explained by the data available currently.

Growth and health are the main dependent variables commonly used to assess efficacy and efficiency of any replacement heifer nutritional programs. Growth in early life in terms of body weight gain has been proposed to be driven more by increasing protein intake than by increasing fat intake (Hill et al., 2008). Health status response in early life to conventional or accelerated nutritional programs has not yet been definitively elucidated from current research, because of the extremely large numbers of calves needed to assess disease outcomes. Therefore, comprehensive economic implications of accelerated nutrition programs are still to be defined.

Growth

Growth performance during the preweaning stage has been observed to be greater for calves fed higher amounts of milk replacers containing greater protein and fat than conventional milk replacer programs. In contrast, calves fed conventional amounts of milk replacers of lower concentrations of protein and fat have shown reduced growth performance in the preweaning

period (Blome et al., 2003; Van Amburgh and Drackley, 2005; Hill et al., 2006a; Hill et al., 2006b; Borderas et al., 2009; Hill et al., 2010).

During the transition period from pre-ruminant to ruminant several physiological and metabolical changes have to occur in the calf in order to become a functional ruminant (Davis and Drackley, 1998). From this perspective, conventional feeding programs give a head-start to the preruminant calf by promoting the consumption of starter earlier in life. Consumption of starter is a key factor in the development of the rumen papillae, which is the main site of absorption of rumen fermentation byproducts. Enhanced feeding programs delay the full development of the rumen in the preweaning period by promoting liquid feed intake over starter (Davis and Drackley, 1998; Borderas et al., 2009). As a result, feed efficiency, ADG, and overall growth often seem to be compromised during the early post-weaning period (Brown et al., 2005; Hill et al., 2006a; Hill et al., 2006b; Stamey, 2008; Borderas et al., 2009; Hill et al., 2010).

Nutritional program effects (conventional or enhanced) after weaning might have critical implications such as overfattening, health problems, poor fertility, reduced subsequent milk production, and others (Drackley, 2005). Detrimental effects on health, fertility, and milk production have been proposed to occur when promoting high growth rates during the post-weaning period (Sejrsen et al., 2000). However, if inadequate nutrition is provided during the rest of the rearing process of the calf, it is more likely that treatment effects achieved by enhanced nutritional programs will be lost in time (Raeth-Knight et al., 2009; Terré et al., 2009). Hence, further research is needed in order to find nutritional thresholds for under- or overfeeding heifers in this stage with an aim not only to decrease the odds of health problems, poor fertility,

and low milk production but also to maintain the nutritional advantages of early enhanced nutritional programs.

Health

Although a considerable body of knowledge has been gained throughout the past decade on effects of early calf nutrition and its impact on health status, the degree of effect of enhanced nutritional programs on health status remains controversial. For instance, it is commonly accepted that feeding milk replacer at higher rates early in life will produce softer fecal consistency (Drackley, 2005; Quigley et al., 2006; Stamey, 2008). However, more fluid feces does not in itself indicate impaired health status in the absence of other signs of disease. Apparent scouring caused by feeding larger amounts of milk replacers more often is an indicator of poor-quality milk replacer (Diaz et al., 2001; Drackley, 2005). Also, it is important to note that whey proteins commonly use in milk replacers do not coagulate in the abomasum; instead, they pass readily into the small intestine for digestion (Davis and Drackley, 1998), which may contribute to a more fluid feces as increased quantities of milk replacers are fed.

According to USDA (2007), the NAHMS reported during 2006 respiratory problems were the second leading cause of death in unweaned heifers and accounted for 22.5% of mortality before weaning. Respiratory problems were the first cause of death in weaned heifers, accounting for 46.5% of total heifer deaths postweaning in dairy farms in the USA. However, total mortality rates are usually much lower in calves after weaning. Therefore, effects of nutrition on respiratory disease seem to be commonly overlooked across studies. Stamey (2008) fed conventional and enhanced nutritional programs to calves from < 1wk old to 10 wk. During

this time mean daily respiratory scores were not different between nutrition programs. Nonnecke et al. (2009) found increased respiratory scores at wk 3 and wk 6 when calves were kept in a cold environment (4.7 °C) compared with those housed in a warm environment (15.5 °C). In the same study, calves under cold environment ate more starter than those in the warm environment, indicating that extra energy is required to maintain comparable performance with that of the calves in the warm environment.

Hoof development and Health

Understanding the normal process of production of hoof horn tissue (keratinization) is critical in order to comprehend how internal and external factors will affect the normal course of the keratinization process. Internal factors encompass any interaction of metabolites and hormones in the keratinization process. There are a number of external factors that can affect hoof development, including temperature, type of floor and bedding system, nutritional program, hoof trimming system, and accidental injuries among others. Following this general information, a summarized review of hoof tissue growth as affected by nutritional approach is provided.

Principles of hoof growth

Hoof tissue growth is a direct product of the process known as keratinization. This consists of the differentiation of epidermal cells at the level of the stratum basale (Figure 1.1) of the coronary band into dead horn cells (Vermunt and Greenough, 1995). Keratins are triple-stranded α-helical polypeptide chains (Steinert and Idler, 1975). A common misconception of

the keratinization process is that it is a degenerative process combined with a drying and posterior apoptosis of epithelial cells; however, keratin is produced through a specific cellular process with the purpose of creating a protein with unique chemical and physical characteristics (Tomlinson et al., 2004).

Dermis and epidermis are the main layers of the skin. The former also is known as "corium" and has a particular function to give support to the epidermis containing a connective tissue layer, blood vessels, and nerves. From the epidermis arise the processes of proliferation and differentiation of the keratinocytes, which ultimately become the cornified ("death cell") cell tissue (Tomlinson et al., 2004). Consequently, the adequate flow of nutrients and certain hormones from the dermis to the epidermis is critical in the continuous assembly of hoof tissue.

The epidermis is divided into stratum basale, stratum spinosum, stratum granulosum, and stratum corneum. As cells move from the stratum basale to the stratum corneum the immature keratinocyte cells undergo several chemical and physical changes, previously referred to as keratinization and cornification. The stratum basale is the main site of cell division. As cells move to the stratum spinosum, keratin synthesis increases rapidly. Here, cells change shape and orientation from upright as in the stratum basale to a horizontal position in the stratum spinosum. The cells also become enlarged, flattened, and polygonal in shape (Mülling, 2000). As cells move through the stratum granulosum, keratin accumulation in the form of basophilic granules and filamentous keratin proteins becomes more evident in the cytoplasm, increasing cell density at the same time. Finally, both types of keratin merge and produce a homogenous mass that fills the cytoplasm of the horn cells (Tomlinson et al., 2004). Towards the end of the stratum corneum, the cytoplasmic organelles of the cells are completely replaced by keratin proteins, leaving a fully developed hoof tissue that eventually migrates down the hoof wall or sole as more

hoof tissue is produced. It is important to emphasize that at the final step of keratinization/cornification the intercellular cementing substance **(ICS)** is synthesized and extruded into the intercellular space. The ICS is a lipid-rich extracellular matrix that, according to Tomlinson et al. (2004), represents the "mortar" and the keratinocytes the "bricks" that will be combine to form a "brick and mortar wall"; thus, ICS primary functions to hold together the keratinocytes.

Nutritional influences on Hoof growth

Blood flow from the dermis into the epidermis via diffusion is critical in order to deliver the adequate amount of nutrients, molecules, and hormones that will promote hoof tissue growth. Therefore, it is important to note that stressors such as cold weather and acidosis, among others, that alter blood flow can impair the extent of any effect of nutrition or feed additives on hoof growth.

Dietary energy effects on hoof tissue growth in terms of energy sources have been studied in depth due to its commonly known implications in lameness problems such as laminitis. A number of studies have focused on the adverse effects of using diets high in readily fermentable carbohydrates or low in effective fiber on rumen acidosis, which is followed by a cascade of events that lead to an increase in endotoxins and histamine that can disrupt blood flow to the hoof. These substances initially result in vasoconstriction but eventually will produce vasodilatation and ischemia in the dermal-epidermal junction of the hoof (Nocek, 1997). The optimal balance of fermentable carbohydrates that allows maximal milk production yet reduces the incidence of laminitis has not been completely determined from previous studies.

Protein requirements, in particular the availability of the amino acids Cys, His, and Met, have been proposed to play an important role in the final step of the keratinization process. For instance, Cys is required to form disulfide bonds in pre-keratinized/cornified hoof tissue, which confer rigidity and high resistance against several proteolytic enzymes (Tomlinson et al., 2004). Also, early lactation cows that commonly express reduced dry matter intake (DMI) may have a limited supply of metabolizable protein, which could diminish protein synthesis by keratinocytes and thus result in low-quality hoof tissue, in turn increasing the predisposition for lameness. In contrast, Vermunt and Greenough (1994) proposed that high protein diets might increase the incidence of laminitis in dairy cattle; however, this hypothesis was not sustained throughout their research literature. The high His content in barley was suggested to be metabolized into histamine or, alternately, that release of histamine was product of an allergic reaction to barley (Maclean, 1966).

Zinc is probably the most widely studied trace mineral due to the large number of biological functions that have been found to be dependent on this mineral. In the case of keratinization, Zn plays a key role in 3 functions: catalytic, structural, and regulatory (Cousins, 2006). Zinc has a catalytic function in keratinization because of its role in enzymes such as RNA nucleotide transferases, RNA polymerase, and alkaline phosphatase, among others. A common feature of these enzymes is that they are Zn metalloenzymes that, as such, depend on Zn as an activator. The structural function of Zn is based on the formation of keratin proteins during the keratinization process; most of this effect is due to zinc-finger proteins that are required in protein-protein interactions (Cousins, 2006). Finally, the regulatory function of Zn on the keratinization process is based on regulation of other hormones, pathways, and enzymes such as

calmodulin, protein kinase C, thyroid hormone binding, and inositol phosphate synthesis (NRC, 2001).

Manganese plays an indirect role in the keratinization process by maintaining adequate cartilage and bone formation, which, in turn, helps to minimize hoof problems. This effect is carried out mainly by the dependency of the enzymes galactotransferase and glycosyltransferase on manganese. These enzymes are needed in the confection of proteoglycan molecules, which are the building blocks of normal cartilage and bone (NRC, 2001).

Copper is important in the activation of enzymes such as cytochrome oxidase, lysyl and thiol oxidases, ceruplasmin, and superoxide dismutase. Cytochrome oxidase is involved in aerobic respiration. Lysyl and thiol oxidases are needed for structural integrity of cells. Ceruplasmin is indispensable for Fe absorption and transport for hemoglobin synthesis, and superoxide dismutase has antioxidant characteristics (NRC, 2001). All of these functions are required for a normal keratinization/cornification processes. For instance, thiol oxidase has the specific function of creating the disulfide bonds between Cys residues in keratin filaments (O'Dell, 1990).

Iron is an essential constituent of the hemoglobin and myoglobin molecules; therefore, it is critical in the transportation of oxygen in the body from the environment to terminal oxidases (Beard, 2006). Therefore, it is critical to have adequate amounts of Fe in order to supply enough oxygen for the keratinization process that requires energy in the form of ATP from the Calvin cycle, where oxygen plus glucose are the main substrates. Also, Fe plays an important role in other molecules; for example, lactoferrin is an iron-binding protein that exhibits broad-spectrum antimicrobial effects.

Lameness

Lameness is the ultimate manifestation of a variety of conditions that may have distinctly different origins (Tomlinson et al., 2004). However, the majority of these conditions are associated, to varying degrees, with lesions within the horny tissues of the hoof (Kempson and Logue, 1993). One of the most common afflictions (especially during lactation in dairy cows) is laminitis, or *Pododermatitis aseptica diffusa* (Vermunt and Greenough, 1994). Since laminitis is a disease with multifactorial etiology, it is considered a generic term for conditions in which the sensitive dermal structures between the pedal bone and the epidermal claw capsule are damaged (Hendry et al., 1997).

Bovine laminitis has been classified into four different forms depending on the severity and duration of the condition, using the terms acute, subacute, chronic, and subclinical. Acute and subacute phases of the disease are characterized by an aseptic inflammation of the corium, which coincides with a systemically sick animal. Chronic laminitis lacks systemic signs and instead signs are localized in the claw. Cows present abnormal horn growth patterns, which affects the normal shape of the claw and results in a rippled appearance of grooves and ridges caused by irregular episodes of horn growth. The subclinical form of laminitis is less well described, certainly is less understood, and usually is not noticed until preventative trimming is performed. The subclinical phase has no systemic signs but rather results in the horn tissue becoming physically softer and discolored, with a waxy appearance (Toussaint, 1989; Vermunt and Greenough, 1994; Donovan et al., 2004).

Predisposing factors for laminitis

Systemic diseases such as mastitis, ketosis, metritis, and rumenitis often occur during the postpartum period and are implicated in the production of toxic substances such as histamine and endotoxin, which then may enter the bloodstream and induce local disturbances or allergic reactions in the micro-circulation of the corium (Maclean, 1971).

Nutrition is certainly a determining factor in the predisposition of laminitis, especially during the transition period. The ideal transition diet should minimize the impact of negative energy balance after parturition, control the DCAD to reduce the incidence of milk fever, retained placenta, and metritis, and prevent overfattening, which can lead to ketosis and fatty liver disease among other possible adverse scenarios. Subsequent to feeding a poor transition diet, release of histamine and endotoxin in the blood can be expected, resulting in greater likelihood of ischemia of the laminae and papillae.

Carbohydrate overload is the principal nutrition-related factor in the development of laminitis (Vermunt and Greenough, 1994). High carbohydrate diets increase rumen bacterial activity, which boosts the production and later accumulation of lactic acid and volatile fatty acids in the rumen, in turn decreasing the rumen pH (Figure 1.2). As ruminal pH decreases below 5, *S. bovis* bacteria are replaced by *Lactobacilli*, at which point progressive decreases in ruminal pH are observed and, as a result, clinical hyperkeratosis and parakeratosis are more likely to occur. This is followed by a further decrease in pH and accumulation of all organic acids with lactate as the predominant and strongest acid (Nocek, 1997). Once the physiological conditions in the rumen have overwhelmed the ability of the microbial population to sequester lactic acid, absorption of lactic acid into the blood stream and systemic acidosis will

occur. From the previous stage until appearance of clinical sings of laminitis, Nocek (1997) proposed a phasic progression of laminitis in four components: initial activation, local mechanical damage, local metabolic insult, and progressive local damage to the bone structure.

The initial activation phase starts with the systemic acidosis increasing digital pulse and total blood flow as a mechanism to regulate pH (Figure 1.3). These insults stimulate the release of endotoxins and histamine, which increase vascular constriction and dilatation; under these conditions the formation of several unphysiological arteriovenous shunts will further increase blood pressure. Subsequently, increased blood pressure will produce seepage through the hoof wall vessels and ultimately lead to edema. The local mechanical damage phase refers to when the edema evolves to ischemia (local anemia) and later tissue hypoxia, which consequently decreases nutrients and oxygen reaching the epidermal cells (Figure 1.3). The local metabolic insult phase is related to a further degree of hypoxia specifically in the stratum germinativum located in the epidermis, which will eventually cause corium degeneration and breakdown of the laminar region or the dermal-epidermal junction (Figure 1.1). During the last phase, a progressive damage of the bone structure occurs. Bone damage is a result of a separation of the stratum germinativum and the corium (i.e., disconnection of dermal-epidermal junction), having the same effect on the dorsal and lateral laminae that support the hoof tissue. Subsequently, the pedal bone shifts in position due to a lack of support in the bottom of the hoof. As a result the pedal bone will compress the soft tissue producing even more hemorrhage, thrombosis, and further enhancement of edema. Ischemia will ultimately create a necrotic area (Nocek, 1997). The proliferation of necrosis, sole ulcers, sole hemorrhages, and appearance of grooves and ridges along the hoof wall are considered clinical signs of laminitis.

Other nutritional factors for predisposition to laminitis include the use of rations low in fiber, specifically in effective fiber, which consequently will reduce ruminal pH. Also the use of diets high in protein has been associated with pathogenesis of laminitis (Maclean, 1971).

Hormonal influences have been linked to laminitis by influencing hoof growth, especially during the postpartum period. For instance, a decrease in insulin sensitivity or concentration in early lactation could compromise production of keratin due to depressed uptake of glucose and amino acids (Hendry et al., 1999). Epidermal growth factor may impact keratin formation and result in formation of inferior horn. Glucocorticoids are thought to have an impact on maturation of keratinocytes through regulation of protein synthesis (Tomlinson et al., 2004); this was confirmed by Hendry et al. (1999) who reported that hydrocortisone inhibited keratin protein synthesis in bovine hoof-tissue explants.

Management factors such as housing, specifically floor type and bedding, must be considered in the etiology of laminitis. For example, Ouweltjes et al. (2009) observed that cows on rubber-topped floors had significantly fewer sole hemorrhages, spent more time standing, and had higher activity. Espejo et al. (2006) implicated a lower prevalence of clinical lameness in Minnesota in comparison to previous years to an increase in use of freestall barns and a decrease in use of tie stalls. Use of sand as bedding conferred positive effects on cleanliness and hoof health in comparison to straw; however, cows preferred straw to sand bedding and laid down longer on straw (Norring et al., 2008).

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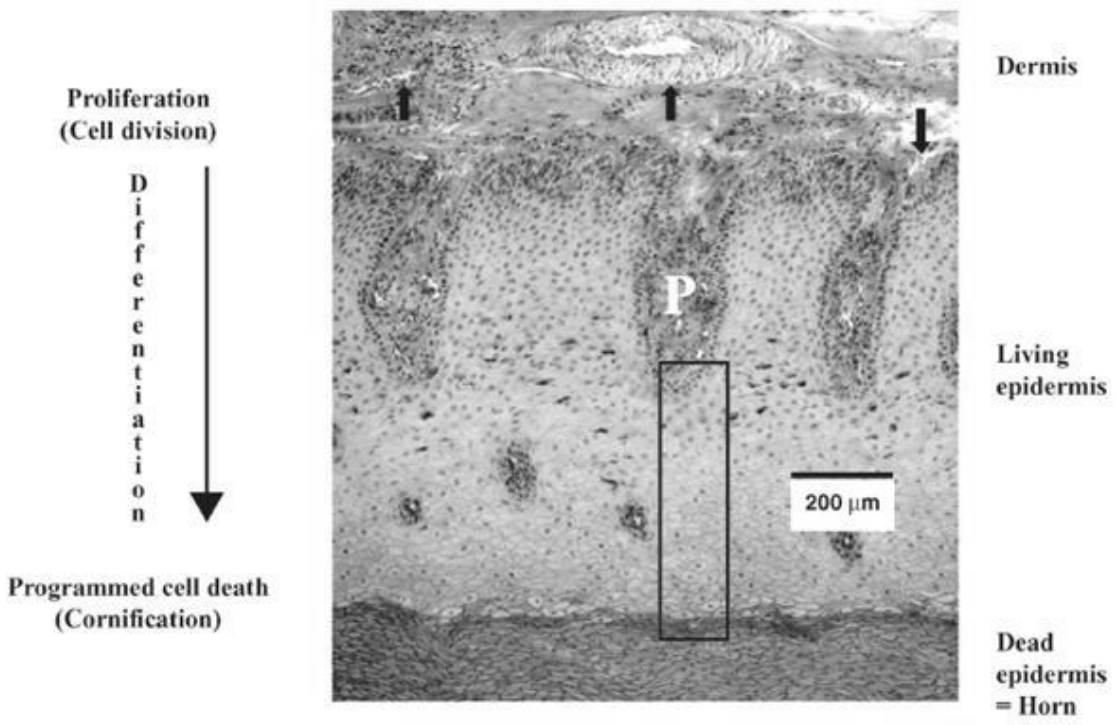

Figure 1.1. Micrograph of the heel region of the bovine hoof. P = Papillae. Source: Adapted from Tomlinson et al. (2004), with permission.

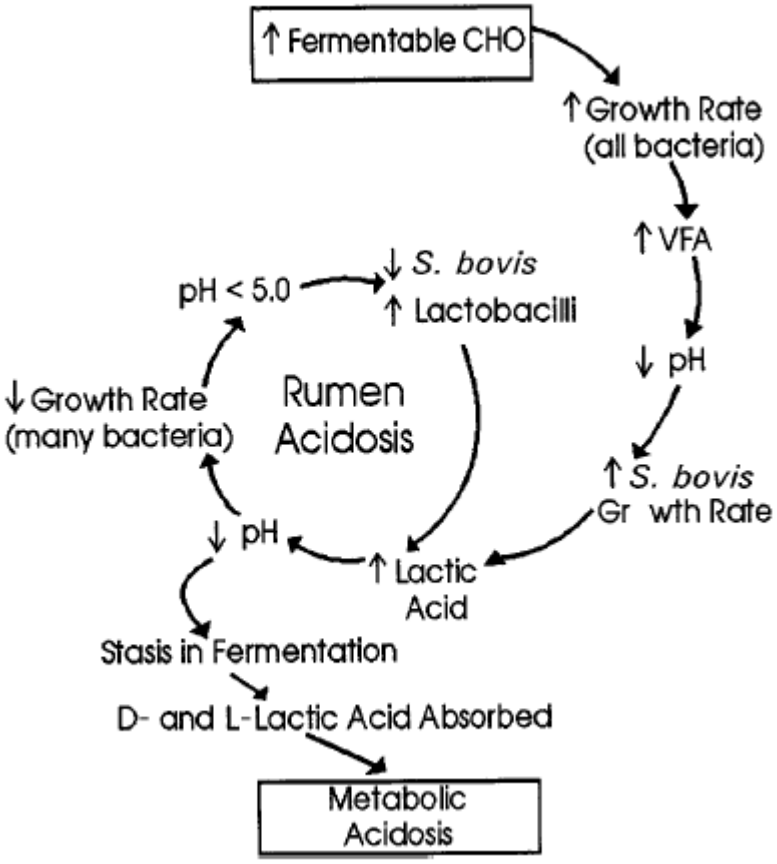

Figure 1.2. Sequence of events associated with the induction of acute ruminal lactic acidosis. CHO = Carbohydrates. Source: Adapted from Nocek (1997), with permission.

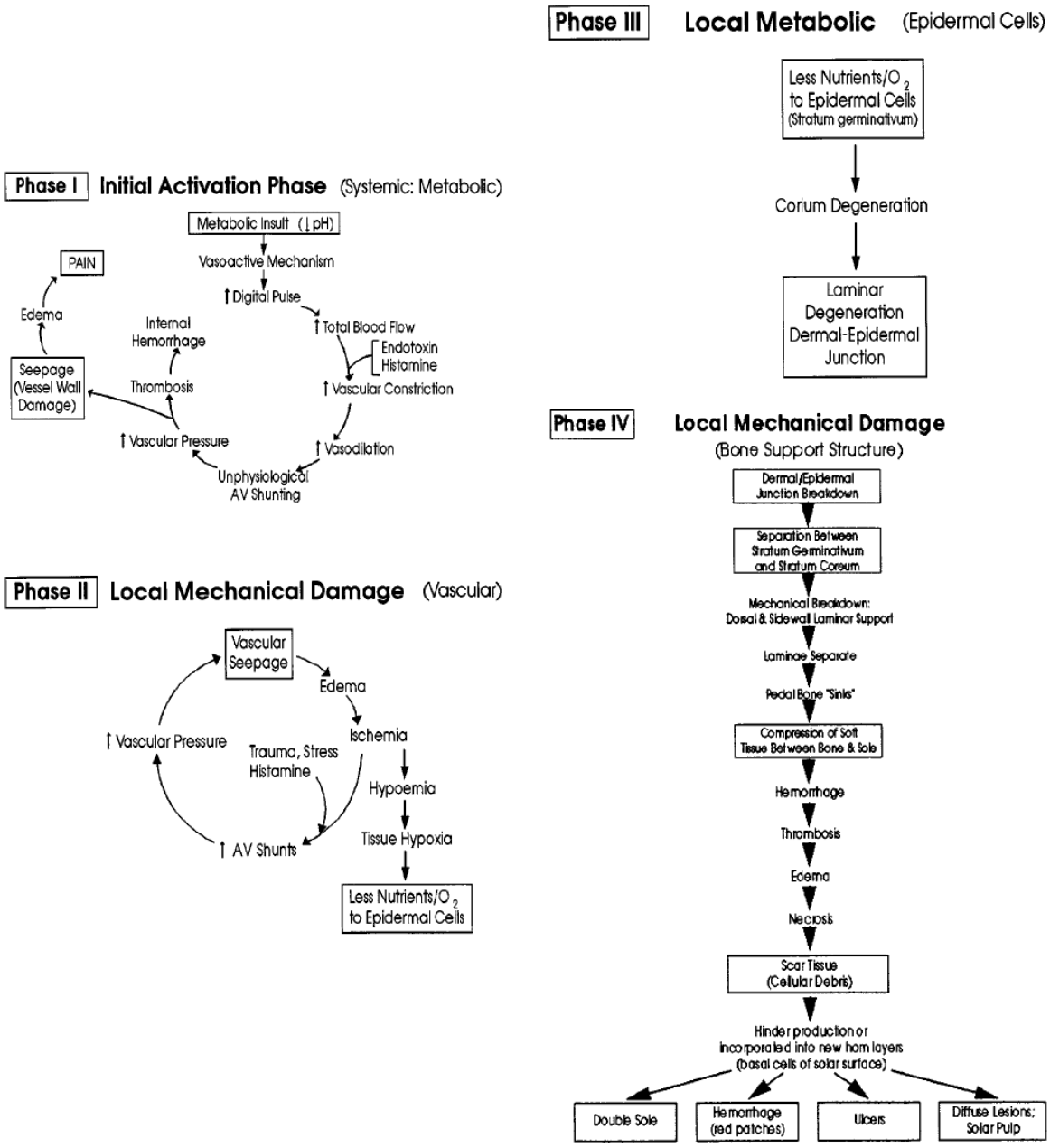

Figure 1.3. The phasic progression of laminitis development. Phase 1, initial activation; phase 2, local mechanical damage; phase 3, local metabolic insult; and phase 4, progressive local damage of the bone structure. AV= Arteriovenous. Source: Adapted from Nocek (1997), with permission.

CHAPTER II

EFFECTS OF SOURCE OF TRACE MINERALS AND PLANE OF NUTRITION ON GROWTH AND HEALTH OF TRANSPORTED DAIRY CALVES

Introduction

Growth and health in newborn dairy calves can be impaired by poor maternal nutrition, difficult calving, and stressors such as transportation and colostrum deprivation; however, these can be mitigated to some extent with quality management and adequate early nutrition. In addition, it has been well documented that feeding more milk replacer of higher protein content (so-called "accelerated" or "enhanced" nutrition") improves early growth performance (Diaz et al., 2001; Blome et al., 2003; Bartlett et al., 2006; Hill et al., 2006a; Hill et al., 2006b). On the other hand, some studies have shown that these accelerated nutrition programs might have a negative impact on health status and, consequently, on overall production cost (e.g., Quigley et al., 2006) for transported calves of questionable colostrum status.

Optimal early growth performance of dairy calves is the main target of accelerated or enhanced early nutrition programs; by increasing DMI of milk replacer calves grow faster and more efficiently early in life. However, common practice has been to encourage early starter consumption by feeding limited amounts of milk replacer to lower feeding cost in conventional nutrition programs. Nevertheless, when choosing a nutritional program it is

important to recognize that growth performance is driven more by increasing protein than by increasing fat intake (Hill et al., 2008).

Negative effects on health status such as increased morbidity (primarily scouring) and use of antibiotics have been attributed to accelerated early nutrition programs (Quigley et al., 2006). However, scouring is not reflected in simply more fluid feces, which should be accepted as a physiological adjustment to increased energy and solids intake. Consequently, improved health status might actually be reflected in less use of antibiotics and electrolytes.

Trace minerals play critical roles in structural development and function of many cellular systems, one of which is the immune system. It is imperative, therefore, that adequate trace mineral status is established at birth and maintained to ensure sufficient stores are available for optimal animal performance and for when animals become disease challenged or stressed. Organic or bioavailable trace minerals have been demonstrated to enhance health status in dairy cattle especially during stress periods such as the periparturient period (Ballantine et al., 2002; Nocek et al., 2006; Griffiths et al., 2007; Siciliano-Jones et al., 2008). Transportation has been found to be an important stress factor that can affect the immunological status by increasing hormones such as cortisol and acute phase proteins that can eventually lead to a decrease in DMI (Stanger et al., 2005). Therefore, transportation was an important stressor component in this trial. Because of the greater nutrient demands for more rapid growth, biological and economic benefits of organic trace minerals might be more pronounced in accelerated early nutrition programs. Therefore, the objective of this study was to determine the effects of source of trace minerals and plane of nutrition on growth and health of transported dairy calves.

Materials and Methods

Experimental Design and Calf Management

All procedures were conducted under protocols approved by the University of Illinois Institutional Animal Care and Use Committee. Ninety Holstein bull calves less than 1 wk old were purchased by a buyer in three groups of 30 to 35 calves from farms in southern Wisconsin and transported to the Nutrition Field Laboratory site at the University of Illinois. Calves were stratified by arrival body weight (BW) and were assigned to one of the four experimental treatments (n = at least 22 per treatment). The treatments for this study (Tables 2.1 and 2.2) were a combination of either conventional (**C**) or accelerated (**A**) plane of nutrition (**PN**) with either inorganic (**I**) or organic (**O**) trace minerals (**TM**) supplemental sources. The experimental design used was a randomized complete-block design with a 2 × 2 factorial arrangement of treatments.

Calves remained at the Field Laboratory facility from arrival through wk 12, and then were transported to the University of Illinois Beef Research Unit for housing through wk 35. Calves were housed in individual hutches from arrival through wk 9, and then were grouped by treatment in super-hutches through wk 12. Calves were housed in groups by original treatment from wk 13 to 20, and then were commingled from wk 21 to 35. During the Field Laboratory phase straw was used as bedding over a base of crushed limestone; calves were housed on rubber-covered concrete slatted floors in pens at the Beef Research Unit.

Calves were castrated 1 wk after arrival.

Calves received diet CI during the week of arrival (wk 0) as a standardization period, and then were switched to assigned treatments at the beginning of wk 1. Calves on C treatments were kept at the same feeding rate (568 g/d); in contrast, calves assigned to A treatments received 810 g/d of powder during wk 1 and 1136 g/d of powder during wk 2 through 5. Milk replacers were reconstituted to 12% solids for all calves during week 0 and 17% and 12% solids to calves fed A and C milk replacers, respectively, until weaning. Fresh starter grain was provided daily and individually by treatment (Table 2.2) from wk 1 through 9. The offered starter was increased 227 g/d when refusal from the previous day was < 136 g. Weaning occurred at the end of wk 6 or wk 7 for C and A treatments, respectively. Weaning procedures were according to the standard recommendations of the milk replacer manufacturer and consisted of lowering the feeding rate to about half (284 and 568 g/d for C and A PN, respectively) during the week preceding weaning. Calves remained in their individual hutches after weaning through wk 9.

After wk 9, calves were grouped by treatment and fed starter grain for ad libitum intake plus a small amount of chopped hay (227 g/d per calf). From wk 13 on, the same general scheme was maintained for groups housed at the Beef Research Unit except that the same grower grain mixture was fed to both C and A PN, with either I or O TM supplemental sources (Table 2.3). For calves fed C, the amount of grower grain mix containing either I or O TM was limited to 3.2 kg/d, with chopped hay provided for ad libitum intake ; whereas, calves fed A were provided grower grain for ad libitum intake but with chopped hay limited to 0.45 kg/d per calf. Nutritional treatments were ended after wk 20, at which time calves from all treatments were changed to a common beef growing diet.

Data Collection

Body conformation measurements including BW, heart girth, body length, withers height, hip height, and hip width were performed after arrival, weekly until wk 10, and then at wk 12, 15, 20, and 35. All measurements were performed by the same person throughout the experiment in order to eliminate variation between observers. A blood sample was obtained by jugular venipuncture on d 1 to determine blood (plasma) protein immediately by refractometry, and IgG concentration by radial-immuno diffusion (RID) method at a later date. Health status (fecal scores and respiratory scores) was recorded daily from wk 0 to 9. For fecal scores a classification from 1 = firm to 4 = liquid was used, and for respiratory scores a scale of normal to dry cough was used (Table 2.4). Individual intakes of milk replacer, starter, and water were measured and recorded daily through wk 7, 9, and 7 respectively (Figures 2.1, 2.2, and 2.3). Then, daily group intakes of starter mix or grower mix and hay were recorded during the super-hutch and Beef Research Unit stages through wk 20. Milk replacer and starter mix were sampled weekly from wk 1 to 7 and wk 1 through 12, respectively. Then, samples were composited in two and three subsamples per replicate for milk replacer and starter mix, correspondingly. In the same fashion weekly samples of grain mix and chopped hay were obtained from wk 13 to 20 during the Beef Research Unit phase and composited in two subsamples (Table 2.3). All subsamples were sent to a commercial laboratory (Dairy One Cooperative Inc., Ithaca, NY), where contents of CP and fat were determined by Leco (AOAC, 1990) and ether extraction (AOAC, 2003). Minerals were determined using a Thermo Jarrell Ash IRIS Advantage HX Inductively Coupled Plasma Radial Spectrometer (Thermo Instrument Systems Inc., Waltham, MA) methods (www.dairyone.com/Forage/Procedures/default.htm).

Calf BW and stature measurements were made at wk 35 to determine any residual effects of early nutrition after receiving the same diet from wk 21 through wk 35.

Statistical Analyses

Daily and weekly data were analyzed using the PROC MIXED procedure of SAS 9.1.3 (SAS Institute Inc., Cary, NC). The model contained the fixed effects of replicate, PN, TM source, the interaction of PN and TM source, time (day or week), and interactions of time with PN and TM source. Calf was considered a random effect and was nested within replicate, PN, and TM source. The covariance structures considered for repeated measures analysis were autoregressive one, compound symmetric, and unstructured; the covariance structure that yielded the lowest Akaike's information criterion was used (Littell et al., 1998). Serum IgG concentration at d 1, BW, body length, heart girth, withers height, hip height, and hip width upon arrival were used as covariates when analyzing the respective measurements. Body growth data were transformed using a square root criterion, in order to achieved homoscedasticity of the residuals. Least squares means were calculated and are presented with standard errors of means (SEM). Significance was declared when $P < 0.05$ and trends at $P < 0.10$.

Results

Nutrient composition of diets

Nutrient composition of milk replacers and starters are shown by treatment in Table 2.1 and Table 2.2, respectively. Measured chemical composition of grain mixes and chopped alfalfa hay used at the beef unit facility can be found in Table 2.3. Milk replacers for C and A PN were

formulated to contain 22% and 28% CP, respectively, on an as-fed basis and actual analyzed CP on a DM basis was 23.5% and 28.9%, respectively. The starters were prepared to contain 18% and 22% CP on as-fed basis for C and A, respectively. Measured CP contents were 23.6% and 28.9% on a DM basis, respectively.

Formulation of milk replacers containing organic TM aimed to provide 50, 50, 10, and 100 ppm of Zn, Mn, Cu, and Fe, respectively; actual chemical analysis indicated Zn, Mn, Cu, and Fe were 58, 53, 11, and 147 ppm correspondingly. Starters with O or I TM contained 144, 116, 27, and 286 ppm and 139, 107, 29, and 284 ppm for Zn, Mn, Cu, and Fe, respectively. In contrast, O and I starters were formulated to contain 105, 91, 21, and 307 ppm and 104, 90, 21, and 306 ppm, correspondingly. Interestingly, Fe and Cu were the most variable TM with more than 3 standard deviations across all treatments and both milk replacers and starters.

Intakes

Mean intakes of milk replacer DM, CP, and ME were greater ($P < 0.0001$) for calves fed the A treatments as expected (Table 2.5). Although TM did not affect DMI, mean intakes of CP were greater ($P = 0.02$) for calves fed O treatments. Calves on A treatments received about twice the DMI of milk replacer compared with calves fed C treatments (Figure 2.1). Intakes of DM, CP, and ME from starter were greater ($P < 0.0001$) for C treatments. Although calves fed C treatments had greater starter DMI due to treatment and weaning effects, A and C calves tended to have similar starter DMI by wk 9 (Figure 2.2). Total DMI did not differ significantly among treatments through wk 9. Total CP intake was greater ($P < 0.05$) for calves fed the A dietary treatments, but total ME intake was not significant among treatments (Table 2.5). A compensation effect on DMI was observed from

wk 1 to wk 9, where C calves tended to balance their low DMI during the first 5 wk with an increased total DMI from wk 5 to wk 9, with the reversed effect for calves fed A diets (Figure 2.3).

Weekly intakes were evaluated in three different periods: liquid (wk 0 to wk 4), weaning (wk 5 to wk 7), and post-weaning (wk 8 to wk 9). During the liquid period total DMI, CP, and ME intakes were greater ($P < 0.0001$) for A treatments. Starter DMI and CP accounted for 27% of total DMI and CP for C treatments; in contrast, starter DMI and CP was 5.5% of total DMI and CP for A treatments. Starter ME differed from the previous pattern, contributing 8.3% and 34.2% of total ME intake for A and C treatments (Figure 2.5).

Throughout the weaning period total DMI and ME intake were greater ($P < 0.05$) for C treatments. However, CP was not significantly different among treatments. Total DMI and CP obtained from starter were increased in both A and C treatments to 59% and 91% of the total, respectively. And, starter ME during the same period contributed to 40.4% and 84.7% of total ME for A and C treatments. Finally, total DMI in the post-weaning period was greater for C treatments. In contrast, total CP and ME remained equal for all treatment effects; greater ME intake for the C treatments (Figure 2.5) did not achieve statistical significance ($P = 0.12$).

Total water, representing free water intake plus water consumed in milk replacer, was greater ($P < 0.0001$) for A treatments in comparison to C treatments during the first 6 wk of age. Free water intake was greater ($P = 0.018$) for A treatments during the first 5 wk of age (Figure 2.4). However, C calves had superior water intake at wk 6 in comparison to a decrease in water intake for A treatments (Figure 2.4). The significant ($P < 0.0001$)

interaction of PN and week was explained by cubic and quartic functions for A and C treatments, respectively, indicating that PN produced different increments across the weeks of study. As designed water intake consumed in the milk replacer was superior ($P < 0.0001$) for calves on A treatments as a result of the greater amount of milk replacer fed in these treatments.

Growth

Calf BW and body conformation measurements did not differ among treatments upon arrival (Table 2.6). Final BW and body conformation measurements at wk 20, except for hip width, were greater ($P < 0.05$) for A treatments. Hip width tended ($P = 0.066$) to be greater for the A treatments than for C. The treatment combination AO was numerically greater for all traits and was significantly greater ($P = 0.05$) than other treatment combinations in final withers height and hip height (Figure 2.6). Residual treatment effects at wk 35 were not significant in all traits, but body length tended ($P = 0.084$) to be greater for A treatments. The interaction of PN and TM was significant ($P < 0.05$) for BW and heart girth, with the treatment combination CO being greater than other treatment combinations. Also, withers height and hip height were greater ($P < 0.05$) for O treatments. The interaction of PN and TM was significant ($P = 0.05$) for BW, body length, withers height, and hip height in which the combination AO was greater than the other treatment combinations; a tendency for the same effect was noted for heart girth and hip width. The interaction of PN and week was significant ($P < 0.05$) for all body conformation characteristics, meaning that the A and C treatments affected growth at different rates over time.

Growth data were then analyzed in four different periods: liquid (wk 0 to wk 4), weaning (wk 5 to wk 7), post-weaning (wk 8 to wk 12), and growing (wk 13 to wk 20). Throughout the liquid period calves fed A treatments were greater than those fed C for BW, heart girth, and hip width. Also, the same trend was observed for body length ($P = 0.10$), withers height ($P = 0.17$), and hip height ($P=0.14$), where differences between A and C treatments did not reach statistical significance. Organic TM resulted in significantly ($P = 0.05$) greater withers height and hip height than the I treatments. The interaction of PN and TM was significant ($P < 0.05$) for all traits except body length and hip width; the treatment combination AO resulted in the greatest ($P < 0.05$) growth.

During the weaning period the A treatments were superior ($P < 0.05$) to C for all growth traits measured. Organic TM tended to have a larger effect than I treatments on withers height ($P = 0.074$) and hip height ($P = 0.067$). However, body length ($P = 0.15$) did not achieve statistical relevance. Significant ($P < 0.05$) interactions of PN and TM were observed on BW, body length, withers height, and hip height, with a tendency for heart girth and hip width. Organic TM had a greater effect than I treatments when applied in combination with the A diet for body length, withers height, and hip height, plus clear tendencies for BW and hip width ($P = 0.065$ and $P = 0.072$, respectively).

Accelerated PN resulted in a greater response for all traits except withers height during the post-weaning period. Organic TM tended to result in a greater withers height ($P=0.066$) and hip height ($P=0.075$). The interaction of PN and TM tended to impact withers height and hip height ($P = 0.084$, and $P = 0.060$, respectively). In the case of BW the interaction effects did not reach statistical relevance ($P = 0.153$). The treatment combination

AO was significantly greater ($P < 0.05$) for withers height and hip height and numerically greater for the other traits.

During the growing period calves fed A were greater ($P < 0.05$) than those fed C for all traits analyzed with the exception of hip width. Comparisons between O and I did not reach statistical significance on withers height and hip height ($P = 0.113$ and $P = 0.159$, respectively). Also, the treatment combination AO was significantly greater than other treatments for hip height and numerically superior for the rest of the traits analyzed.

Mean ADG and G:F were greater ($P<0.05$) for calves fed A treatments (Table 2.6). Throughout the liquid period ADG was greater ($P < 0.0001$) for A, and the interaction of PN and TM was significant ($P < 0.05$), with the treatment AO greater than the other treatments (Figure 2.7). The same pattern was observed on G:F with the exception of a tendency for AO to be greater ($P=0.061$) than other treatments (Figure 2.8). Treatments did not affect ADG at weaning and post-weaning. A trend ($P=0.069$) for greater ADG for A treatments was observed during the growing period, whereas C treatments actually decreased ADG. Accelerated PN had greater ($P < 0.0001$) G:F during the weaning period; however, treatment effects were not significant post-weaning. Analysis of residual treatment effects at wk 35 showed a tendency for greater ($P = 0.082$) ADG for calves previously fed O TM. The interaction of PN and TM was significant ($P < 0.05$), with treatment CO greater than other treatments.

Health

Calves fed A treatments presented more ($P < 0.0001$) fluid feces than those fed C diets (Table 2.7). Also, there was a significant interaction of PN and TM in which the AI treatment

had a greater fecal score than other treatments. The same pattern was observed with days scoured where A treatments had on average 3 more ($P < 0.05$) days with fluid feces than calves fed C treatments. In addition, the AI treatment had ≥ 1.6 more days with scours than other treatments. As a consequence, use of electrolytes to maintain fluid balance was greater ($P = 0.05$) for AI than other treatments. Mean respiratory score was not different ($P > 0.5$) among treatments.

Analyzed IgG concentrations did not differ significantly among treatments at the beginning of the experiment. However, calves assigned to A diets tended (P=0.12) to have a higher IgG upon arrival; therefore, as described, the initial IgG was used as a covariate in statistical analyses. During the study mortality was lower for the A diets and lowest for the combination of AO, although the analysis (PROC GENMOD) did not declare this significant ($P = 0.67$). In addition, the survival curve (Figure 2.9) illustrates the stress period early in life between wk 1 and 2, where all treatments experienced a fall in the percentage survival.

Discussion

The results from several experiments previously published concur with results of the present study. Increased feeding rates of milk replacer reduced consumption of starter throughout the milk feeding period, as observed in other studies where greater amounts of milk or milk replacer were fed (Jenny et al., 1982; Huber et al., 1984; Jasper and Weary, 2002; Brown et al., 2005; Quigley et al., 2006; Hill et al., 2006b, 2008; Borderas et al., 2009). Also, free water intake was increased by A treatments; in contrast, Quigley et al. (2006) and Bartlett et al. (2006) did not report any effect of feeding rates on water intake. However, water intake increased sharply at wk 6 for C treatments, surpassing that of A treatments, which is consistent with a rapid

increase in dry feed intake due to weaning effects (Davis and Drackley 1998; NRC, 2001). Introduction of calves to alfalfa hay after 9 wk might have added some variation to our model in terms of equal amount of trace minerals to be delivered. Alfalfa hay presents different concentrations of nutrients depending on the vegetative stage to harvest and harvesting, and storage procedures (Balde et al., 1993).

Calves fed A diets had increased overall growth, ADG, and G:F, which agrees with previous experiments (Diaz et al., 2001; Jasper and Weary, 2002; Blome et al., 2003; Bartlett et al., 2006). Organic TM increased animal stature, reflected in greater withers height and hip height, particularly when fed in the A diet. Although feces were more fluid for calves fed A diets, health status was not disturbed. In fact, increased water intake by A might have helped to maintain fluid balance, which did not happen in the Quigley et al (2006) and Bartlett et al. (2006) studies. Although use of electrolytes to maintain fluid balance was greater, the fecal score for calves fed the AI diet was numerically greater than that for AO-fed calves.

Interestingly, residual dietary effects at wk 35 on BW and ADG were significantly greater ($P < 0.05$) for calves previously fed O diets, with heart girth and body length traits also showing the same tendencies.

Conclusions

Our results described the responses of dairy calves to increased feeding rate of milk replacer and use of organic or inorganic trace minerals supplemental sources. Accelerated PN treatments increased overall growth, ADG, and G:F through wk 20. Organic TM increased growth when supplemented to the A diet but not when supplemented to the C diet. Moreover, a

presumed synergistic interaction occurred between A and O by means of increasing the nutritional value of each other. Organic TM increased animal stature as reflected in greater withers and hip height. Although feces were more fluid, calves fed A diets did not have impaired health status, perhaps due in part to an increase in free water and consequently increased total water intake through 5 wk of age contributing to homeostatic fluid balance. Residual effects of organic TM were present at wk 35 on BW and ADG, with the same tendency for heart girth and body length.

Implications

Our results indicated that feeding organic TM had a greater benefit for calves fed in accelerated feeding programs than those receiving conventional limited amounts of milk replacer. Promoting free water intake early in life may ameliorate the negative effects of increased fluid feces in enhanced nutritional programs, and allow increased starter intake.

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Table 2.1. Chemical composition of milk replacer by treatment.

	Treatment[1]				
Nutrient[2]	CI	CO	AI	AO	SEM[3]
DM, %	93.1	93.5	94.1	94.5	0.53
CP, % of DM	23.5	23.7	28.7	29.2	0.24
Fat, % of DM	21.6	21.4	21.3	19.7	0.64
ME, Mcal/kg	3.41	3.41	3.26	3.17	0.03
Ca, % of DM	1.09	1.08	0.79	0.8	0.01
P, % of DM	0.87	0.86	0.86	0.86	0.01
Mg, % of DM	0.15	0.15	0.14	0.14	0.002
K, % of DM	2.74	2.73	2.52	2.53	0.03
Na, % of DM	1.13	1.13	1.14	1.13	0.03
S, % of DM	0.35	0.34	0.43	0.43	0.01
Fe, ppm	126	168	101	126	17
Zn, ppm	50	57	50	59	5
Cu, ppm	13	10	10	11	3
Mn, ppm	52	54	48	52	3
Mb, ppm	1	1	1	1	1

[1] Treatments: CI = conventional PN inorganic TM; CO = conventional PN organic TM; AI = accelerated PN inorganic TM; AO = accelerated PN organic TM. Inorganic trace minerals (Zn, Mn, Cu, Co) in sulfate forms. Organic trace minerals (Zn, Mn, Cu, Co) supplied by ZINPRO, MANPRO, Cu-PLEX, and COPRO.

Table 2.1 (cont.)

[2] Feed samples were taken weekly from wk 1 to 8 and composited in two subsamples for each replicate.

[3] SEM = standard error of the mean.

Table 2.2. Chemical composition of starter grain by treatment.

	Treatment[1]				
Nutrient[2]	CI	CO	AI	AO	SEM[3]
DM, %	87.1	87.4	87.4	87.5	0.26
CP, % of DM	22.4	21.8	25.4	26.9	1.00
ADF, % of DM	9.9	10.1	9.2	10.4	0.66
NDF, % of DM	19.6	19.4	15.8	16.2	0.59
Lignin, % of DM	2.5	2.7	2.5	2.4	0.28
NFC, % of DM	45.9	47.2	47	44.9	1.55
Fat, % of DM	3.6	3.4	3.2	2.8	0.15
ME, Mcal/kg	3.02	3.01	3.10	3.08	0.03
Ca, % of DM	1.34	1.32	1.27	1.35	0.08
P, % of DM	0.64	0.60	0.59	0.60	0.02
Mg, % of DM	0.24	0.23	0.22	0.23	0.01
K, % of DM	1.26	1.28	1.38	1.46	0.05
Na, % of DM	1.56	1.44	1.49	1.61	0.08
S, % of DM	0.31	0.34	0.31	0.31	0.02
Fe, ppm	276.6	280.7	290.7	292.9	11.21
Zn, ppm	155	147.7	122.7	140.4	13.09
Cu, ppm	29.6	27.7	28.1	26.2	1.77
Mn, ppm	109.8	129.7	104.1	101.5	8.93
Mb, ppm	0.9	0.8	0.8	1.00	0.28
Co, added ppm	1	1	1	1	

Table 2.2 (cont.)

Cu, added ppm	12	12	12	12
Fe, added ppm	20	20	20	20
Zn, added ppm	69	70	70	70

[1]Treatments: CI = conventional PN inorganic TM; CO = conventional PN organic TM; AI = accelerated PN inorganic TM; AO = accelerated PN organic TM. Inorganic trace minerals (Zn, Mn, Cu, Co) in sulfate forms. Organic trace minerals (Zn, Mn, Cu, Co) supplied by ZINPRO, MANPRO, Cu-PLEX, and COPRO.

[2] Feed samples were taken weekly from wk 1 to 8 and composited in two subsamples for each replicate.

[3] SEM = standard error of the mean.

Table 2.3. Chemical composition of grower grain mix and chopped hay at the beef research unit by treatment.

	Grain mix[1]			Chopped	
Nutrient[2]	Inorganic	Organic	SEM	hay	SEM[3]
DM, %	88.3	88.0	0.33	90.9	0.35
CP, % of DM	24.5	22.6	1.07	12.6	0.37
ADF, % of DM	12.1	11.5	0.63	45	0.80
NDF, % of DM	21.4	19.6	0.50	59.8	0.90
TDN, % of DM	79	79.3	0.89	57	0.93
NE_m, Mcal/kg	0.88	0.89	0.01	0.50	0.01
NE_g, Mcal/kg	0.59	0.60	0.01	0.25	0.01
Ca, % of DM	1.76	1.51	0.14	0.82	0.06
P, % of DM	0.92	0.81	0.05	0.23	0.01
Mg, % of DM	0.295	0.27	0.01	0.21	0.01
K, % of DM	1.54	1.40	0.11	2.27	0.12
Na, % of DM	0.45	0.39	0.03	0.03	0.002
S, % of DM	0.32	0.29	0.02	0.14	0.01
Fe, ppm	421	381	40.69	148	20.3
Zn, ppm	179	173	14.05	21	0.81
Cu, ppm	34	29	2.51	8	0.70
Mn, ppm	144	140	6.75	28	1.11
Mb, ppm	1.05	0.73	0.18	0.35	0.12
RFV				84	1.82

Table 2.3 (cont.)

Co, added ppm	1	1
Cu, added ppm	12	12
Fe, added ppm	20	20
Zn, added ppm	70	70

[1]Grain mix: Inorganic trace minerals (Zn, Mn, Cu, Co) in sulfate forms. Organic trace minerals (Zn, Mn, Cu, Co) supplied by ZINPRO, MANPRO, Cu-PLEX, and COPRO.

[2]Feed samples were taken weekly from wk 13 to 20 and composited in two subsamples for each replicate.

[3]SEM= Standard error of the mean.

Table 2.4. Health scores classification.

Fecal scores		Respiratory scores	
Score	Description	Score	Description
1	Firm, well formed (not hard)	1	Normal
2	Soft, pudding like	2	Runny nose
3	Runny, pancake batter	3	Heavy breathing
4	Liquid, splatters	4	Cough moist
		5	Cough dry

Table 2.5. Mean intakes of dry matter (DM), crude protein (CP), and metabolizable energy (ME) from milk replacer and starter, and mean free water intake from wk 1 to wk 9.

Variable	Treatment[1]					Contrast[2]		
	CI	CO	AI	AO	SEM[3]	I vs. O	A vs. C	PN*TM[4]
Milk replacer DM, g	516	514	947	990	17.96	0.599	<.0001	0.22
Starter DM, g	1365	1318	819	873	91	0.969	<.0001	0.576
Total DM, g	1683	1634	1535	1593	92	0.965	0.298	0.552
Milk replacer CP, g	113	114	264	273	61	0.021	<.0001	0.019
Starter CP, g	315	293	211	228	23	0.92	0.0003	0.371
Total CP, g	390	368	416	439	23	0.995	0.032	0.32
Milk replacer ME, Mcal	1.764	1.754	3.191	3.143	0.737	0.131	<.0001	0.284
Starter ME, Mcal	4.095	3.966	2.565	2.72	0.278	0.916	<.0001	0.604
Total ME, Mcal	5.182	5.042	4.91	5.002	0.279	0.93	0.569	0.673
Free water intake, kg/d	2.24	2.23	2.70	2.88	0.65	0.716	0.018	0.692

[1]Treatments: CI = conventional PN inorganic TM; CO = conventional PN organic TM; AI = accelerated PN inorganic TM; AO = accelerated PN organic TM. Inorganic trace minerals (Zn, Mn, Cu, Co) in sulfate forms. Organic trace minerals (Zn, Mn, Cu, Co) supplied by ZINPRO, MANPRO, Cu-PLEX, and COPRO.

Table 2.5 (cont.)

[2]Contrast statements between inorganic and organic TM and accelerated and conventional PN.

[3]Largest standard error mean SEM of all treatments.

[4]Interaction between inorganic and organic TM and accelerated and conventional PN.

Table 2.6. Initial and final body weight (BW), final body conformation measurements at wk 20, average daily gains (ADG), and feed efficiency.

	Treatments[1]					Contrast[2]		Interaction
Variable	CI	CO	AI	AO	SEM[3]	I vs. O	A vs. C	PN*TM[4]
Initial BW, kg	44.7	44.5	44.9	44.6	0.88	0.723	0.779	0.995
Final BW, kg	177.3	170.1	186.1	188.3	8.30	0.567	< 0.05	0.287
Heart girth, cm	128.2	127.5	131.1	132.1	1.80	0.890	<0.001	0.377
Length, cm	101.3	101.3	103.4	105.4	0.97	0.279	< 0.05	0.291
Withers height, cm	105.1	105.6	106.0	108.0	1.09	0.094	<0.05	0.293
Hip height, cm	109.5	109.6	110.1	112.1	1.05	0.138	< 0.05	0.193
Hip width, cm	30.1	29.8	30.3	30.7	0.55	0.824	0.066	0.332
ADG, kg/d[5]	0.83	0.80	0.88	0.91	0.04	0.966	0.037	0.315
G:F, g/g[6]	0.45	0.41	0.49	0.55	0.03	0.754	<.0001	<0.05

[1]Treatments: CI = conventional PN inorganic TM; CO = conventional PN organic TM; AI = accelerated PN inorganic TM; AO = accelerated PN organic TM. Inorganic trace minerals (Zn, Mn, Cu, Co) in sulfate forms. Organic trace minerals (Zn, Mn, Cu, Co) supplied by ZINPRO, MANPRO, Cu-PLEX, and COPRO.

[2]Contrast statements between inorganic and organic TM and accelerated and conventional PN.

[3]Largest standard error mean SEM of all treatments.

[4]Interaction between TM and PN treatment effects.

Table 2.6 (cont.)

[5] ADG was estimated from wk 1 to wk 20

[6] Gain to feed efficiency was estimated from wk 1 to wk 20

Table 2.7. Mortality characteristics, arrival mean IgG, daily fecal scores, days with scours, electrolytes, and antibiotics from wk 1 through wk 9.

Variable	Treatments[1]					Contrast[2]		
	CI	CO	AI	AO	SEM[3]	I vs. O	A vs. C	PN*TM[4]
Mortality, %	13	13	9.1	4.5	—	0.295	0.718	0.670
Age at mortality, day	26	17.7	8.5	13	—			
IgG, mg/dL	1074	1193	1420	1409	212	0.766	0.123	0.719
Fecal Scores	1.35	1.53	1.71	1.60	0.06	0.545	<0.001	<0.05
Scours, days	5	9	11	10	4	0.300	0.001	<0.05
Electrolytes, days	1	2	3	1	1.9	0.228	0.107	<0.05
Antibiotics[5], days	2	1	2	2	0.49	0.219	0.459	0.646

[1]Treatments: CI = conventional PN inorganic TM; CO = conventional PN organic TM; AI = accelerated PN inorganic TM; AO = accelerated PN organic TM. Inorganic trace minerals (Zn, Mn, Cu, Co) in sulfate forms. Organic trace minerals (Zn, Mn, Cu, Co) supplied by ZINPRO, MANPRO, Cu-PLEX, and COPRO.

[2]Contrast statements between inorganic and organic TM and accelerated and conventional PN.

[3]Largest standard error mean SEM of all treatments.

[4]Interaction between TM and PN treatment effects.

[5]Number of days treated for respiratory problems.

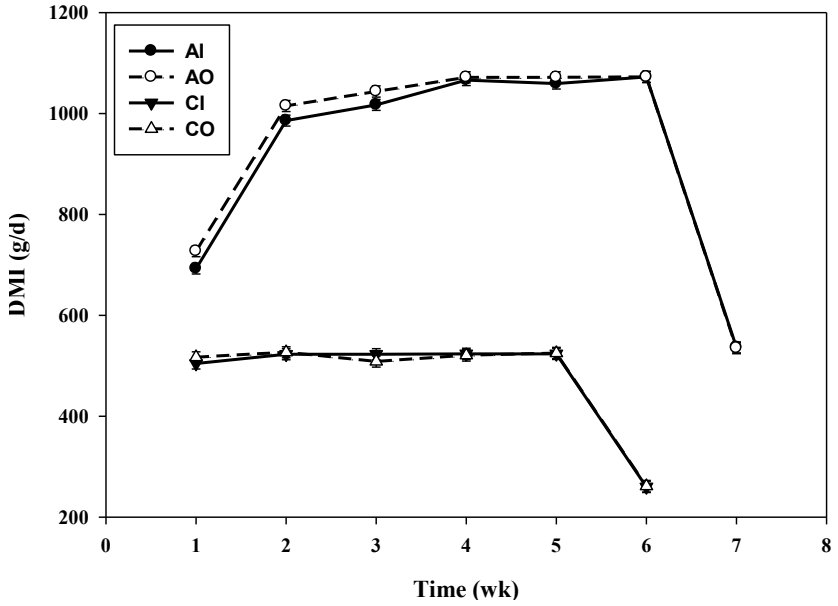

Figure 2.1. Mean daily intakes of milk replacer from wk 1 to wk 7 for calves fed conventional PN and inorganic TM (CI), conventional PN and organic TM (CO), accelerated PN and inorganic TM (AI), accelerated PN and organic TM (AO).

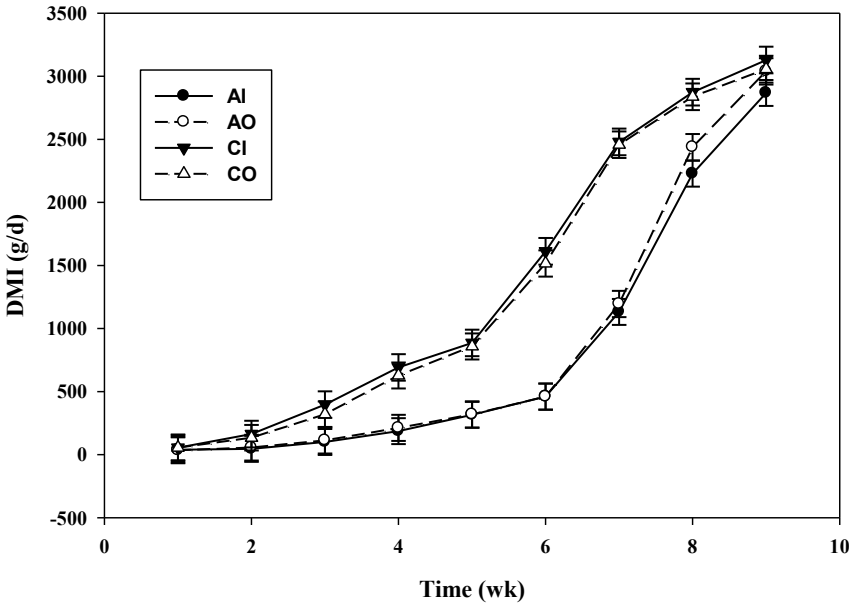

Figure 2.2. Mean daily intakes of starter from wk 1 to wk 9 for calves fed conventional PN and inorganic TM (CI), conventional PN and organic TM (CO), accelerated PN and inorganic TM (AI), accelerated PN and organic TM (AO).

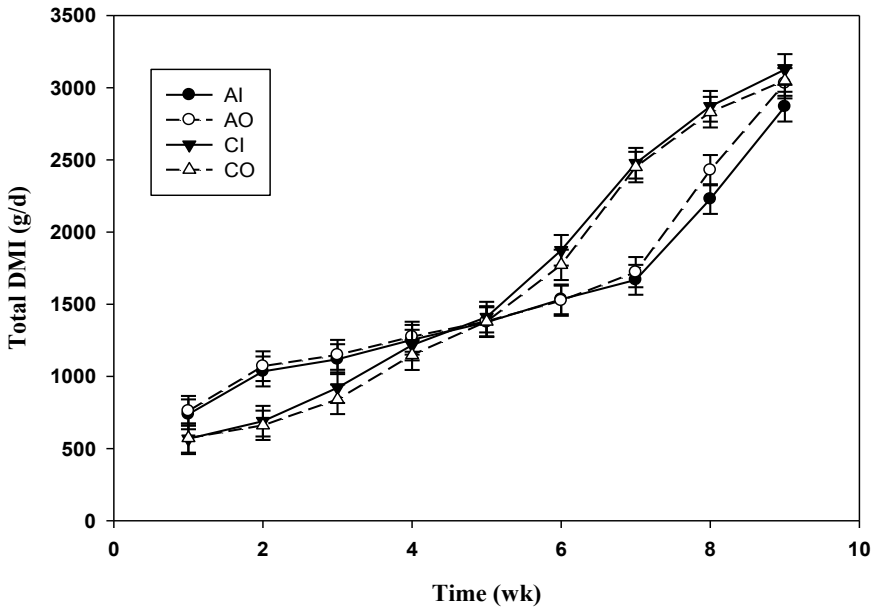

Figure 2.3. Mean total DMI from wk 1 to wk 9 for calves fed conventional PN and inorganic TM (CI), conventional PN and organic TM (CO), accelerated PN and inorganic TM (AI), accelerated PN and organic TM (AO).

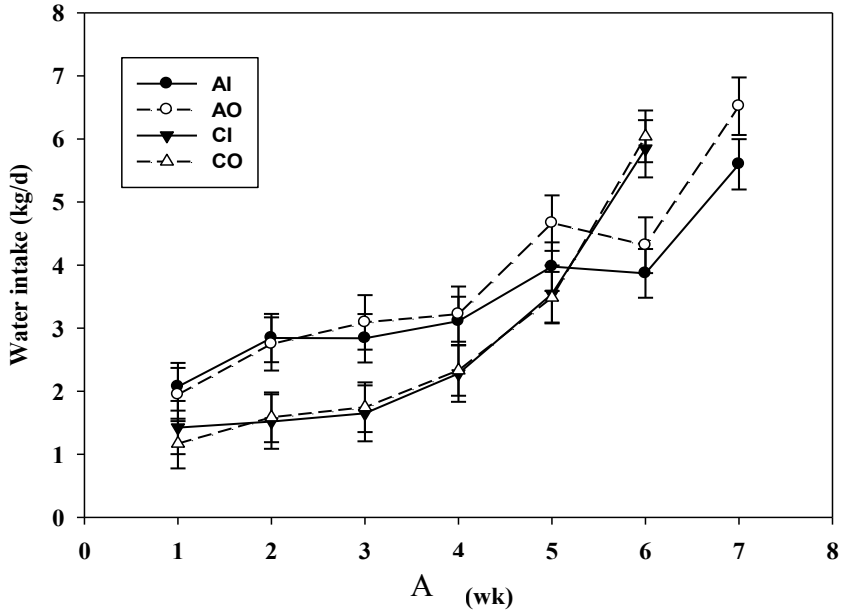

Figure 2.4. Mean daily intakes of water from wk 1 to wk 9 for calves fed conventional PN and inorganic TM (CI), conventional PN and organic TM (CO), accelerated PN and inorganic TM (AI), accelerated PN and organic TM (AO).

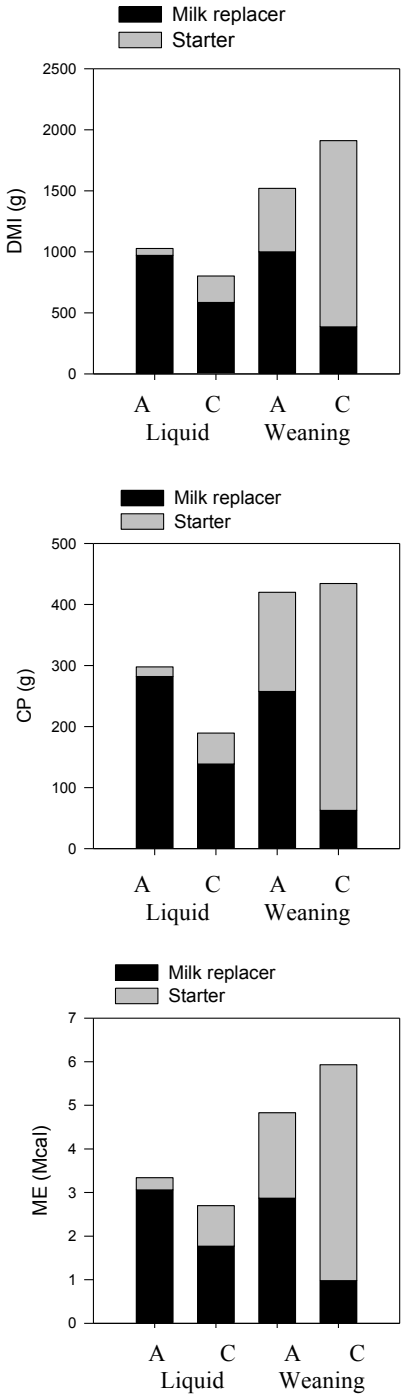

Figure 2.5. Mean contribution of milk replacer or starter by PN treatment effect on total DM (Panel A), total CP (Panel B), total ME (Panel C) during the liquid and weaning period from wk 0 to wk 4 and wk 5 to wk 7, respectively. A=accelerated and C=conventional.

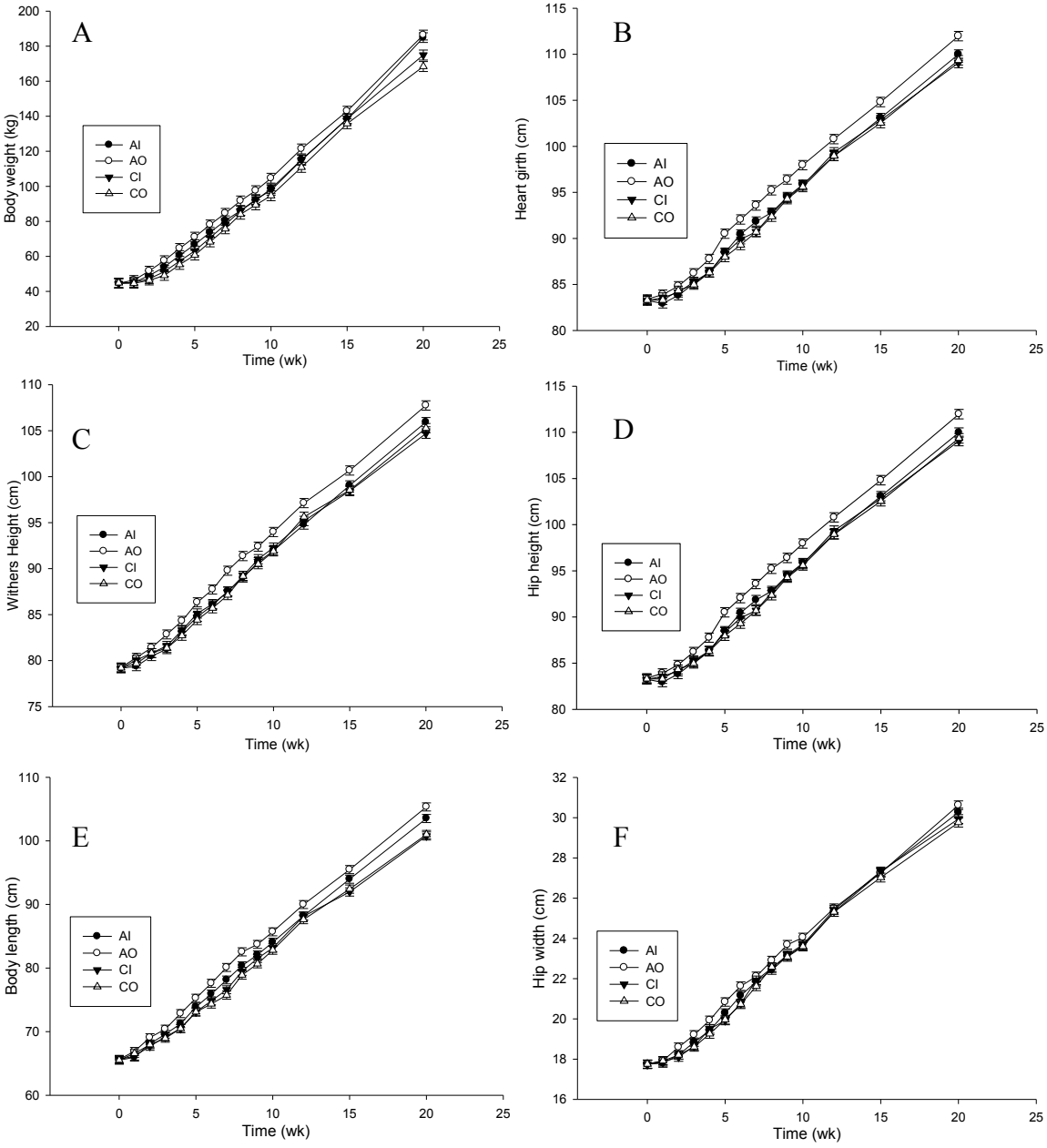

Figure 2.6. Mean weekly body weight (BW;(panel A), heart girth (HG; panel B), withers height (WH; panel C), hip height (HH; panel D), body length (BL; panel E), and hip width (HW; panel F) from wk 0 to wk 20 for calves fed conventional PN and inorganic TM (CI), conventional PN and organic TM (CO), accelerated PN and inorganic TM (AI), accelerated PN and organic TM (AO).

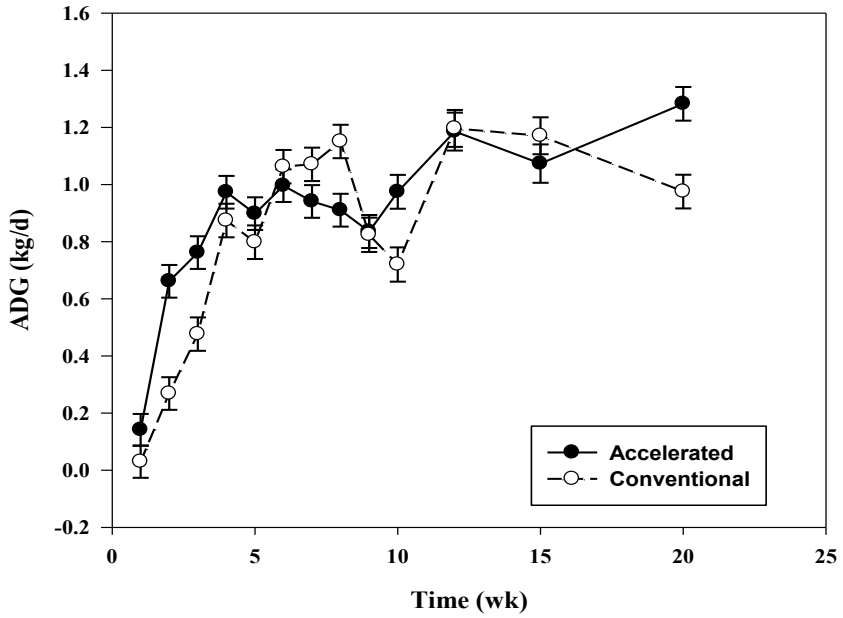

Figure 2.7. Average daily gain from wk 1 to wk 20 for calves fed conventional or accelerated PN.

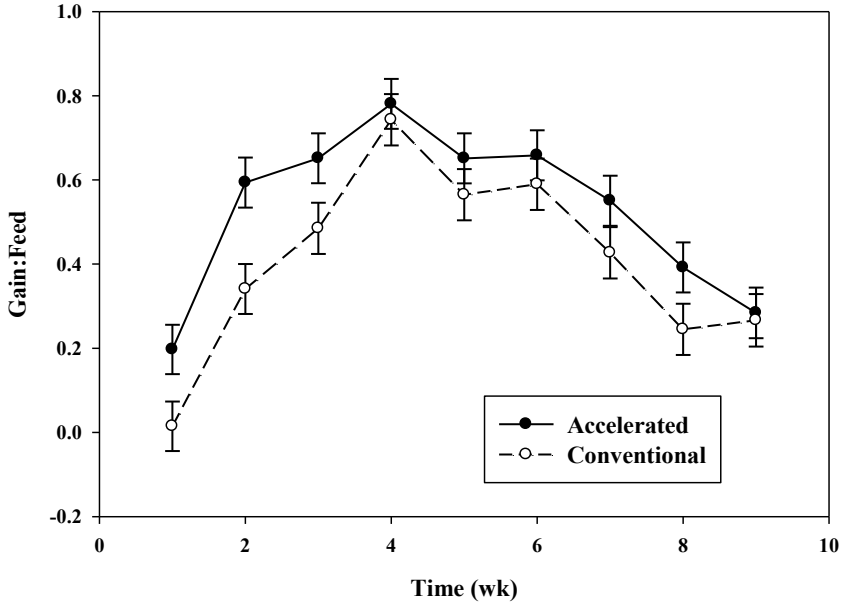

Figure 2.8. Mean gain: feed ratio from wk 1 to wk 9 for calves fed conventional or accelerated PN.

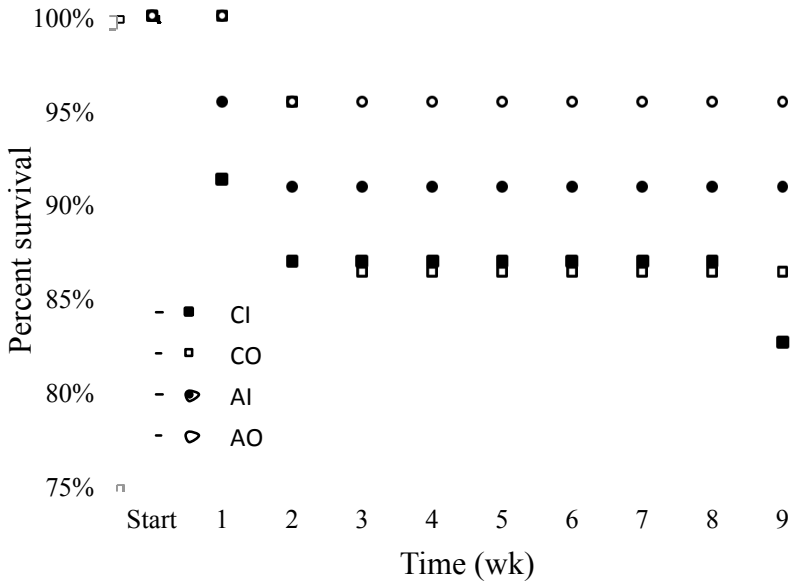

Figure 2.9. Survival curve from wk 1 to wk 9 for calves fed conventional PN and inorganic TM (CI), conventional PN and organic TM (CO), accelerated PN and inorganic TM (AI), accelerated PN and organic TM (AO).

CHAPTER III

EFFECTS OF SOURCE OF TRACE MINERALS AND PLANE OF NUTRITION ON HOOF DEVELOPMENT IN TRANSPORTED DAIRY CALVES

Introduction

Lameness in dairy cattle has been well described in several studies as the result of different conditions that lead to a common locomotion problem. Lameness is a major direct cause of economic losses in the dairy industry (Hendry et al., 1997; Green et al., 2002; Donovan et al., 2004; Neveux et al., 2006). In addition, indirect economic losses from decreased production, increased treatment cost, prevalence, reduced fertility, and ultimately increased involuntary culling are related to lameness (Neveux et al., 2006). Moreover, an increased concern for animal welfare by consumers has brought lameness to the attention of researchers, producers, and the dairy industry in general and has highlighted the need to find solutions to this problem through prevention and improved treatments.

Trace minerals (Zn, Mn, Cu, Se, Fe, and Co) are fundamental in the keratinization and cornification processes of hooves. They activate enzymes such as RNA nucleotide transferases, RNA polymerase, and protein kinase C, which affect cell differentiation and increase production of keratin proteins. Trace minerals activate Cu/Zn superoxide dismutase and glutathione peroxidase, which are essential enzymes for protection of lipids from peroxidation. One such

susceptible material is the lipid-rich extracellular matrix, the so-called ICS that is responsible for holding together the squames (keratinized cells) during the cornification process (Tomlinson et al., 2004). In addition, there are close interrelationships among these TM; thus, synergistic interactions have been observed in several studies (Nocek et al., 2000; Ballantine et al., 2002; Nocek et al., 2006; Griffiths et al., 2007) showing positive effects on claw integrity and lactation performance. Inadequate ingestion, absorption, storage, and vascular supply of TM impair normal production and assembly of keratinized cells that determine hoof health status. Absorption and storage might be the most common issues in supply of adequate amounts of TM because of the problems of enterocyte transporter saturation, depressed absorption by competition from other minerals, and poor storage capacity. Low DMI during stressed periods also might limit adequacy of TM status.

Production of replacement heifers in the dairy industry, especially in the USA, has become a subject of much interest because producers are more aware of the economic significance of an inefficient replacement growing program. Losses accrue through increased feeding cost for each month of delayed pregnancy, low first lactation production due to overfattening or undergrowth, reproductive problems, and early death or culling. Therefore, a specific replacement nutrition program is required in which the main objective is to prepare the future cow for optimal production and to withstand stress periods. One aspect of such a program might be the need to prepare heifers to minimize lameness problems during future lactations by achieving adequate hoof development in early life. Use of organic TM might impact the vascular irrigation of claw tissue, providing a more dynamic and reactive chorium that can adapt to environmental and metabolic changes. These improvements might become more observable

over time, presumably as result of some additive effect of using organic TM in diets that facilitate rapid growth (Drendel et al., 2005). Whether such effects of TM source would vary depending on the rates of early growth is an important factor to determine. Therefore, the objective of this study was to evaluate the effects of source of TM and plane of nutrition on hoof development in transported dairy calves.

Materials and Methods

Calves and Treatments

All procedures were conducted under protocols approved by the University of Illinois Institutional Animal Care and Use Committee and were described in detail in Chapter II. Three groups (replicates) of Holstein calves < 1 wk old were purchased from farms in southern Wisconsin and transported via livestock trailer to the Nutrition Field Laboratory site at the University of Illinois. Upon arrival, each calf was bottle-fed 3.8 L of control milk replacer (conventional plane of nutrition plus inorganic TM). All calves were administrated 2 mL TSV-2 ® (Pfizer, Inc., New York, NY) intranasally, 1.0 mL of Mu-Se (Shering-Plough Animal Health Corp., Union, NJ) via intramuscular injection, and a prophylactic injection of 1.5 mL of Excede (Pfizer, Inc., New York, NY) via subcutaneous injection. During the first week rectal temperatures were recorded daily and navels were coated daily with Povidone iodine (Henry Schein, Inc., Melville, NY). All calves were castrated 1 wk after arrival.

Dietary treatments were a combination of either conventional (**C**) or accelerated (**A**) plane of nutrition (**PN**) with either inorganic (**I**) or organic (**O**) trace minerals (**TM**) supplemental sources. The experiment was conducted as a randomized complete-block design with a 2 × 2 factorial arrangement of treatments.

Housing

Calves remained at the Nutrition Field Laboratory facility from arrival through wk 12 and then were transported to the University of Illinois Beef Research Unit for housing through wk 35. Calves were housed in individual hutches from arrival through wk 9 and then were grouped by treatment in super-hutches through wk 12. Straw was used as bedding over a base of crushed limestone. At the Beef Research Unit, calves were housed in pens by original treatment group from wk 13 to 20, and then were commingled and fed a common diet from wk 21 to 35. Pens contained rubber-covered concrete slatted floors.

Feeding

Calves received the CI milk replacer during the week of arrival (wk 0) as a standardization period, and then were switched to assigned treatments at the beginning of wk 1. Calves on the C treatments were kept at the same feeding rate (568 g/d); in contrast, calves assigned to A feeding treatments received increased amounts of milk replacer (810 g/d during wk 1; 1136 g/d during wk 2 to 5). Milk replacers were reconstituted to 12% solids for all calves during week 0 and 17% and 12% solids to calves fed A and C milk replacers, respectively until weaning. Fresh starter grain was offered daily and individually by treatment from wk 1 through 9. The amount of starter provided was increased 227 g/d when refusal from previous day was <

136 g. Calves were weaned at the end of wk 6 and 7 for C and A treatments, respectively. Weaning procedures were according to the standard recommendations of the milk replacer manufacturer and consisted of decreasing the feeding rate by one-half (284 and 568 g/d for conventional and accelerated PN, respectively) during the week preceding weaning. Calves remained in their individual hutches after weaning through wk 9.

After wk 9 calves were grouped by treatment and fed starter grain for ad libitum intake plus a small amount of chopped hay (227 g/d per calf). From wk 13 on, the same general scheme was maintained for groups housed at the beef unit except that only one grower grain mixture was fed to both C and A calves, which contained either I or O sources of TM. For C calves, the amount of grower grain mix containing either source of TM was limited to 3.2 kg/d with chopped hay provided for ad libitum intake, whereas the A calves were provided grower grain for ad libitum intake but with chopped hay limited to 0.45 kg/d per calf. Milk replacer and starter mix were sampled weekly from wk 1 to 7 and wk 1 through 12, respectively. Then, samples were composited in two and three subsamples per replicate for milk replacer and starter mix, correspondingly. In the same fashion weekly samples of grain mix and chopped hay were obtained from wk 13 to 20 during the Beef Research Unit phase and composited in two subsamples. All subsamples were sent to a commercial laboratory (Dairy One Cooperative Inc., Ithaca, NY), where contents of CP, fat, and minerals were determined by Leco (AOAC, 1990), ether extraction (AOAC, 2003), and Thermo Jarrell Ash IRIS Advantage HX Inductively Coupled Plasma Radial Spectrometer (Thermo Instrument Systems Inc., Waltham, MA) methods, respectively (www.dairyone.com/Forage/Procedures/default.htm).

Nutritional treatments were ended after wk 20, at which time calves from all treatments were changed to the same growing diet. The period from wk 21 to wk 35 on a common diet was to determine whether any residual effects of early life treatments persisted through that time.

Hoof development, growth, and wear

Evaluation of hoof development was by visual inspection and claw measurement with a graduated rule from the coronary band to the end of the hoof wall and from coronary band to the groove line, yielding the variables claw length (**CL**) and groove length (**GL**), respectively (Figure 3.1). Hoof development was evaluated upon arrival and then at wk 5, 10, 15, 20, and 35 after treatment initiation. All evaluations were performed by the same person throughout the experiment in order to eliminate variation between observers. Groove length was not measurable until wk 10 due to the poorly defined groove line before that time. Hoof evaluation at wk 35 was an assessment of residual treatment effects because calves from all early treatments were fed a common growing diet after wk 20.

Growth and wear of hooves was analyzed from wk 0 to wk 20 and for intervals between evaluation times based on an adaptation of the procedures of Hahn et al. (1986). Overall growth was assumed to be GL at wk 20 and wear was calculated as $wear = CL0 - (CL20 - GL20)$ where *CL*20 and *CL*0 are the claw lengths at wk 20 and 0, respectively. Growth and wear were also calculated at 5-wk intervals during the 20 wk of nutritional treatments. During interval 1 (wk 0 to 5) we assumed negligible wear because calves spent most of the time lying down. Hoof growth was calculated as $growth = CL5 - CL0$ where *CL*5 and *CL*0 are the claw lengths at wk 5 and 0, respectively. Then, for interval 2 (wk 5 to wk 10), growth was calculated as $growth =$

$GL10 - (CL5 - CL0)$ where GL10 is the groove length at wk 10. Subsequently, wear was determined as $wear = CL0 - (CL10 - GL10)$, assuming that the length below the groove $(CL10 - GL10)$ will be smaller than *CL*0, due to wear effects. Growth and wear for intervals 3 (wk 10 to wk 15) and 4 (wk 15 to wk 20) were obtained in the same way, e.g., $growth = GL15 - GL10$ and $wear = (CL10 - GL10) - (CL15 - GL15)$.

Two additional variables were calculated: net growth and ratio, which were calculated as $net\ growth = growth - wear$ and $ratio = growth/wear$, for the overall trial (wk 0 to wk 20) and for intermediate intervals 2, 3, and 4. Residual effects were analyzed by growth and wear from wk 20 to wk 35. Residual growth was calculated as $growth = GL35 - GL20$ and wear was calculated as $wear = (CL20 - GL20) - (CL35 - GL35)$.

Statistical analysis

Overall CL and GL plus net growth and wear by phase were analyzed using the PROC MIXED procedure of SAS (version 9.1.3; SAS Institute Inc., Cary, NC). The model contained the effects of PN, TM, the interaction of PN and TM, time (week or interval), and interactions of time with PN and TM. Week with the subject of claw nested within calf were used as a repeated statement. The covariance structure that yielded the lowest Akaike's information criterion was used for repeated measurements. Claw length at arrival was used as a covariate in the models. Least squares means were calculated and are presented with standard errors of means (SEM). Significance was declared when $P < 0.05$ and trends at $P < 0.10$.

Results

Trace Mineral Composition of Diets

Milk replacers were formulated to provide 50, 50, 10, and 100 ppm of Zn, Mn, Cu, and Fe, respectively; actual chemical analysis indicated Zn, Mn, Cu, and Fe were on average 58, 53, 11, and 147 ppm (Table 3.1). Organic TM and inorganic TM starters were formulated to contain 105, 91, 21, and 307 ppm and 104, 90, 21, and 306 ppm of Zn, Mn, Cu, and Fe, respectively. In contrast, chemical analysis indicated that O and I starters contained 144, 116, 27, and 286 ppm and 139, 107, 29, and 284 ppm (Table 3.2).

Analysis of mean daily intakes of Zn showed that calves fed C had greater ($P < 0.0001$) intakes (Figure 3.2.a). The interaction of PN and TM was significant ($P = 0.018$), which was caused by the calves fed CI tending ($P = 0.078$) to have greater Zn intakes. Daily Mn intakes were greater ($P < 0.0001$) for C treatments than for A and greater ($P = 0.003$) for O than I treatments (Figure 3.2.b). Calves fed C diets had greater ($P < 0.0001$) daily intakes of Cu and Fe than did calves fed A diets (Figures 3.2.c and 3.2.d, respectively). These effects agree with the more rapid increase in total DMI of calves fed C after about 4 wk of age due to greater intakes of starter compared with calves fed A (Figure 3.3).

Initial and Final Hoof Measures

Claw length upon arrival was greater for medial and rear claws ($P < 0.001$) than for lateral and front claws, respectively (Figure 3.4). Also, rear medial claws were longer than the other claws (claws 6 and 7 in Figure 3.4). Because of differences at arrival, initial claw length

was used as a covariate for all models. Final claw length at wk 20 was greater for calves fed O diets ($P < 0.0001$), but final groove length was greater ($P = 0.0003$) for calves fed A diets.

Repeated measurements of hooves

Claw length was greater for calves fed O diets ($P = 0.006$) and tended ($P = 0.06$) to be greater for calves fed A. These effects were largely driven by the increment in length from wk 11 until wk 15 for calves fed the AO treatment combination and then for calves fed CO from wk 16 to the end of the treatment period (Figure 3.5). Rear claws had greater ($P < 0.0001$) claw length then front claws. Analysis by location showed that the significant TM effect was driven by the effect of O on the rear and medial claws more than the front and lateral claws (Table 3.3).

Groove length was greater for calves fed the AO treatment combination ($P = 0.008$); consequently, PN was superior ($P < 0.0001$) for A treatments; although O was numerically greater than I this effect was not statistically ($P = 0.12$) significant (Figure 3.6). Accelerated PN had a consistent effect of increasing groove length regardless of claw location (Table 3.3).

Growth and Wear

Overall hoof growth presented a numerically larger effect for calves fed A diets to be greater than those fed C, however this differences did not achieve statistical significance ($P = 0.15$). Also, the treatment combination AO was numerically larger than the other treatments (Figure 3.7), which was similar to the previously discussed results. Accelerated PN resulted in a greater ($P = 0.005$) overall wear than the C treatments. Although the interaction of PN and TM

was not significant ($P = 0.14$), the CO treatment combination tended ($P = 0.097$) to result in lower wear in comparison to other treatments (Figure 3.8).

Treatments did not affect net growth for the overall trial from wk 0 to wk 20; however, the C diets resulted in a greater ($P < 0.05$) ratio growth/wear in comparison to A treatments (Figure 3.9). Also, the interaction of PN and TM did not reach statistical significance ($P = 0.13$), but the CO treatment was greater ($P < 0.05$) than the other combination treatments. Based on the previously presented results, these altered ratios must be interpreted as a result of lower wear rather than higher growth. The ratio for rear medial claws (claws 6 and 7 in Figure 3.4) was greater ($P = 0.05$) in comparison to other claws (Figure 3.10).

Analysis of growth by location established a particular effect of PN ($P = 0.057$) on the rear claw position (Table 3.3). Consequently, most of the increment observed in growth of A versus C treatments in Figure 3.7 was a result of the specific effect of PN on the rear claws. This effect is also evident (Table 3.4) where differences in claw position (1&4 = front, lateral; 2&3 = front, medial; 5&8 = rear, lateral ; 6&7 = rear, medial) were greater for calved fed the A treatments in comparison to the C diets . Analysis of wear by location showed an effect of PN to increase hoof wear by increased delivery of nutrients by the A feeding programs (Table 3.3). In addition, the interaction of PN and TM was significant ($P = 0.018$) only for the rear claw position (Table 3.3), suggesting that changing from inorganic to organic TM in a conventional feeding program will produce a lower effect (Figure 3.8), with a more marked effect in the rear claws. Also, the interaction PN and TM is more clear for the AO treatment combination in Table 3.5 where AO was greater ($P = 0.014$) than other treatments at every measurement for rear

medial claws versus other claws; however, this effect was lesser when front claws were compared to each other (2&3 vs 1&4).

Hoof growth by interval is shown in Figure 3.11. Growth during interval 1 (wk 0 to wk 5) was minimum (0.3mm/wk), however through interval 2 (wk 5 to wk 10) growth increased dramatically (2.5mm/wk). Growth declined linearly during intervals 3 (wk 10 to wk 15) and 4 (wk 15 to wk 20).

Growth by interval time was greater ($P = 0.005$) for A treatments, specifically during the second interval (Figure 3.12.a). Wear by interval time was affected ($P < 0.0001$) by PN. Accelerated PN produced a mild decrease in wear, while in contrast the C treatments showed a quadratic response over time (Figure 3.12.b).

Ratio growth/wear by interval time was significantly ($P < 0.0001$) greater for C treatments and greater ($P = 0.002$) for O diets (Figure 3.12.c). Net growth (as $net\ growth = growth - wear$) by interval time was greater ($P = 0.044$) for effect diets.

Residual hoof growth at wk 35 was not affected by original treatments (Figure 3.13). Residual wear at wk 35 also was not affected by treatments (Figure 3.14). However, the interaction of PN and TM was significant ($P = 0.049$), denoting an increment in wear by calves that had previously received the CO diet compared with those fed CI. On the other hand, calves previously fed AO tended to show decreased wear in comparison to those fed AI. Effects on wear were opposite for PN and TM.

Discussion

Chemical analysis showed that contents of Zn, Mn, Cu, and Fe present in the milk replacer (Figure 3.4) were similar to those suggested in the NRC (2001), as was the objective in formulation. In contrast, TM concentrations in the starter grain were greater (Figure 3.15) than recommendations by the NRC (2001). However, Jenkins and Hidiroglou (1991) did not observe any impairment of calf performance by feeding milk replacer supplemented with Zn and Mn ≤ 700 and ≤ 1000 ppm, respectively. Similarly, milk replacer containing ≤ 2000 ppm of Fe did not decrease weight gains, DMI, or feed efficiency (Jenkins and Hidiroglou, 1987). According to Jenkins and Kramer (1989) milk replacer supplemented with Cu at rates < 1000 ppm did not cause reduced weight gains or feed efficiencies. Thus, no adverse effects of the higher starter TM contents would be expected.

Data available on the nutritional effects of trace minerals and level of nutrition on horn tissue development specifically during early life of mammals is limited, and the extent of these nutritional effects on newborn dairy calves has not been presented before. Differences in claw length upon arrival were consistent with findings from Keller et al. (2008) where the distal limb bone length was measured in 1-d-old calves by radiography. Keller et al. (2008) found that lateral claws were longer than medial claws ($P < 0.01$). Their results concurred with those of Hahn et al. (1986) who recorded hoof growth and wear for 20 mo in primiparous and multiparous Holstein cows and found the same pattern of increased growth and wear of rear claws. Nevertheless, in the study by Hahn et al. (1984), medial front and lateral rear claws were longer than respective opposing claws. In the present study medial claws were longer than lateral regardless of front or rear position. This suggests a possible change in weight distribution

from calves after birth to mature cows. According to Neveux et al. (2006) cows shift most of their weight towards the front legs. Furthermore, Toussaint (1989) argued that, due to a wide variation in weight bearing, the lateral rear claws grow faster than the medial rear claws, which have more constant weight bearing and, consequently, more uniform wear.

The rapid increase in claw growth during the second interval (wk 6 to 10) of the treatment period suggests that something in the calves' environment or physiology changed markedly during this period. A number of factors could lead to this response, including increased thermoregulation, development of rumination, increased DMI, and decreased concentrations of hormones known to be negative for hoof growth such as glucocorticoids, epidermal growth factor, insulin, and glucose (Tomlinson et al., 2004). For instance, Hendry et al. (1999) observed that hydrocortisone inhibited keratin protein synthesis in bovine hoof tissue explants. In addition, the same researchers found that epidermal growth factor may impair keratin formation due to its potent mitogenic and anti-differentiative effects. Hendry et al. (1999) reported that insulin binding was detected in both epidermal and dermal layers of isolated bovine hoof tissue; after an incubation of 24 h in the presence of insulin protein and DNA synthesis were increased in the same tissue samples. The researchers proposed that as insulin resistance builds up during stress periods such as in early lactation cows, detrimental claw-horn keratin production will occur due to depressed uptake of glucose and amino acids.

Weekly growth from wk 0 to wk 20 was 1.48 mm and 1.50 mm for front and rear claws, respectively, which were slightly lower than data of Hahn et al. (1986) who reported 1.51 mm and 1.64 mm for front and rear claws, respectively. However, growth was not uniform over the entire interval. Specifically, very little growth (0.3 mm/wk) occurred during interval 1 between

wk 0 and wk 5 (Figure 3.11), in contrast to a peak in growth during interval 2 that was about 1 mm/wk greater than reported by Hahn et al. (1986). Therefore, interval 2 (from wk 5 to wk 10) should be considered a very important period to understand and analyze corium development, its interactions with environmental and dietary factors, and its consequences in lameness susceptibility later in life.

Calves fed A diets consumed less TM overall, regardless of TM source (Figure 3.2) because of the much greater TM content of starter compared with milk replacer and the greater total intake of starter by calves fed C. Nevertheless, hoof growth by interval was greater (P = 0.005) for calves fed the AO diet, confirming that enhanced nutritional programs allow a greater effect of TM on hoof growth by increasing the biological value of TM.

More desirable flooring conditions during the Beef Research Unit period (from wk 12 to wk 20) might have diminished differences among treatments. According to Ouweltjes et al. (2009) cows housed on rubber-covered slatted floors had lower incidence of sole hemorrhages, had larger claws, spent more time standing, and had higher activity then cows on concrete floors. Offer et al. (2003) suggested that feeding a dry straw-based diet is advantageous to claw health in contrast to feeding fermented grass silage-based diets during rearing and subsequent lactation.

No hoof lesions were observed in any calves at any time throughout the experiment, suggesting that energy levels and sources on the diet did not induce severe acidic conditions or that any sign of clinical hoof lesion might be accumulative and will be present later in life. However, the present study confirmed results from Momcilovic et al. (2000), who attempted to induce laminitis by feeding calves (17 wk of age) with diets higher in energy (81%

vs 71% TDN) and higher in protein (20% vs 15% CP) than in controls. In comparison, in our experiment the I and O grain mixes offered throughout the Beef Research Unit period contained 79.0% vs 79.3% TDN and 24.5% vs 22.6% CP, respectively (Table 3.4). Regardless, neither diet fed by Momcilovic et al. (2000) produced signs of laminitis even though the 81% TDN diets resulted in lower ruminal pH and increased D- and L-lactate concentrations.

Conclusions

Our results describe the response of young calves to increased feeding rates of milk replacer, increased energy and protein content in milk replacer and starter grain, and use of O or I sources of supplemental TM in milk replacer and starter. Calves are born with uneven claw lengths; rear and medial claws were longer than front and lateral claws. Hoof growth was most rapid for all treatment groups during wk 5 to 10. Organic TM increased claw length after wk 10 in comparison to I TM, which might be associated with an accumulative effect of O supplementation. Organic TM increased groove length when fed in the A diet but not in the C diet. Organic TM sources tended to decrease net wear when supplemented to the C diet. Rear claws were more susceptible to treatment effects on growth and wear. The A dietary programs had a greater effect to increase overall hoof growth; in contrast, O diets had a greater effect on reducing overall hoof wear.

Implications

Rapidly increased hoof growth during wk 5 to wk 10 suggests a consolidation of corium cells during this time. Therefore, it is important to provide enough nutrients during this

period to ensure a healthy hoof development. Accelerated nutritional programs supplemented with O sources of supplemental TM promoted greater hoof growth, while O acted to decrease overall hoof wear compared with I sources.

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Table 3.1. Chemical composition of milk replacer by treatment.

	Treatment[1]				
Nutrient[2]	CI	CO	AI	AO	SEM[3]
DM, %	93.1	93.5	94.1	94.5	0.53
CP, % of DM	23.5	23.7	28.7	29.2	0.24
Fat, % of DM	21.6	21.4	21.3	19.7	0.64
ME, Mcal/kg	3.41	3.41	3.26	3.17	0.03
Ca, % of DM	1.09	1.08	0.79	0.8	0.01
P, % of DM	0.87	0.86	0.86	0.86	0.01
Mg, % of DM	0.15	0.15	0.14	0.14	0.002
K, % of DM	2.74	2.73	2.52	2.53	0.03
Na, % of DM	1.13	1.13	1.14	1.13	0.03
S, % of DM	0.35	0.34	0.43	0.43	0.01
Fe, ppm	126	168	101	126	17
Zn, ppm	50	57	50	59	5
Cu, ppm	13	10	10	11	3
Mn, ppm	52	54	48	52	3
Mb, ppm	1	1	1	1	1

[1] Treatments: CI = conventional PN inorganic TM; CO = conventional PN organic TM; AI = accelerated PN inorganic TM; AO = accelerated PN organic TM. Inorganic trace minerals (Zn, Mn, Cu, Co) in sulfate forms. Organic trace minerals (Zn, Mn, Cu, Co) supplied by ZINPRO, MANPRO, Cu-PLEX, and COPRO.

Table 3.1 (cont.)

[2] Feed samples were taken weekly from wk 1 to 8 and composited in two subsamples for each replicate.

[3] SEM = standard error of the mean.

Table 3.2. Chemical composition of starter grain by treatment.

	Treatment[1]				
Nutrient[2]	CI	CO	AI	AO	SEM[3]
DM, %	87.1	87.4	87.4	87.5	0.26
CP, % of DM	22.4	21.8	25.4	26.9	1.00
ADF, % of DM	9.9	10.1	9.2	10.4	0.66
NDF, % of DM	19.6	19.4	15.8	16.2	0.59
Lignin, % of DM	2.5	2.7	2.5	2.4	0.28
NFC, % of DM	45.9	47.2	47	44.9	1.55
Fat, % of DM	3.6	3.4	3.2	2.8	0.15
ME, Mcal/kg	3.02	3.01	3.10	3.08	0.03
Ca, % of DM	1.34	1.32	1.27	1.35	0.08
P, % of DM	0.64	0.60	0.59	0.60	0.02
Mg, % of DM	0.24	0.23	0.22	0.23	0.01
K, % of DM	1.26	1.28	1.38	1.46	0.05
Na, % of DM	1.56	1.44	1.49	1.61	0.08
S, % of DM	0.31	0.34	0.31	0.31	0.02
Fe, ppm	276.6	280.7	290.7	292.9	11.21
Zn, ppm	155	147.7	122.7	140.4	13.09
Cu, ppm	29.6	27.7	28.1	26.2	1.77
Mn, ppm	109.8	129.7	104.1	101.5	8.93
Mb, ppm	0.9	0.8	0.8	1.00	0.28

Table 3.2 (cont.)

Co, added ppm	1	1	1	1
Cu, added ppm	12	12	12	12
Fe, added ppm	20	20	20	20
Zn, added ppm	69	70	70	70

[1]Treatments: CI = conventional PN inorganic TM; CO = conventional PN organic TM; AI = accelerated PN inorganic TM; AO = accelerated PN organic TM. Inorganic trace minerals (Zn, Mn, Cu, Co) in sulfate forms. Organic trace minerals (Zn, Mn, Cu, Co) supplied by ZINPRO, MANPRO, Cu-PLEX, and COPRO.

[2] Feed samples were taken weekly from wk 1 to 8 and composited in two subsamples for each replicate.

[3] SEM = standard error of the mean.

Table 3.3. Least squares means for claw length, groove length, net growth, and net wear by location and treatment effect from 0 to wk 20.

Claw length							
Claw	Plane of Nutrition		*P*	Trace Minerals		*P*	Interaction
Position	Accelerated	Conventional		Inorganic	Organic		PN*TM
Front	48.51	48.25	0.106	48.23	48.52	0.071	0.581
Rear	50.28	50.04	0.154	49.94	50.39	0.007	0.471
Lateral	49.00	48.69	0.094	48.68	49.01	0.069	0.843
Medial	49.79	49.60	0.303	49.50	49.89	0.032	0.977
Groove length							
Claw	Plane of Nutrition		*P*	Trace Minerals		*P*	Interaction
Position	Accelerated	Conventional		Inorganic	Organic		PN*TM
Front	22.43	21.60	0.002	21.89	22.14	0.348	0.272
Rear	22.83	21.61	<.0001	22.03	22.41	0.156	0.082
Lateral	22.54	21.51	0.0001	21.85	22.20	0.191	0.159
Medial	22.72	21.70	0.0001	22.07	22.35	0.295	0.152
Net growth							
Claw	Plane of Nutrition		*P*	Trace Minerals		*P*	Interaction
Position	Accelerated	Conventional		Inorganic	Organic		PN*TM
Front	29.93	29.37	0.40	29.48	29.81	0.62	0.55
Rear	30.55	29.28	0.057	29.76	30.07	0.64	0.56
Lateral	30.22	29.26	0.14	29.56	29.92	0.58	0.38
Medial	30.25	29.39	0.18	29.68	29.96	0.66	0.75
Net wear							
Claw	Plane of Nutrition		*P*	Trace Minerals		*P*	Interaction
Position	Accelerated	Conventional		Inorganic	Organic		PN*TM
Front	18.17	16.90	0.025	17.68	17.39	0.613	0.71
Rear	15.88	14.44	0.004	15.43	14.90	0.283	0.018
Lateral	17.44	16.00	0.005	16.90	16.54	0.486	0.063

Table 3.3 (cont.)

Medial	16.61	15.34	0.008	16.20	15.75	0.330	0.36

Table 3.4. Least square means estimates for net growth comparisons among claw position (1&4 = front, lateral; 2&3 = front, medial; 5&8 = rear, lateral; 6&7 = rear, medial) and PN treatment effect from 0 to 20 wk.

Contrast		Estimate	SEM	P
Claw Position	PN and TM	-0.9	0.47	0.072
6&7 vs 5&8	Conventional	-0.2	0.46	0.636
2&3 vs 1&4	Accelerated	0.9	0.48	0.055
2&3 vs 1&4	Conventional	0.7	0.46	0.148
6&7 vs 2&3	Accelerated	0.3	0.47	0.515
6&7 vs 2&3	Conventional	-0.6	0.46	0.182
6&7 vs 1&4	Accelerated	1.2	0.47	0.01
6&7 vs 1&4	Conventional	0.05	0.46	0.912
5&8 vs 2&3	Accelerated	1.2	0.47	0.01
5&8 vs 2&3	Conventional	-0.4	0.46	0.388
5&8 vs 1&4	Accelerated	2.1	0.47	<0.0001
5&8 vs 1&4	Conventional	0.3	0.46	0.56

Table 3.5. Least mean squares of contrast for net wear comparisons among claw position (1&4 = front, lateral; 2&3 = front, medial; 5&8 = rear, lateral; 6&7 = rear, medial) and PN and TM treatment effects from 0 to 20 wk.

Contrast		P
Claw Position	PN and TM	
6&7 vs 5&8	AO vs CI	0.001
6&7 vs 5&8	AO vs CO	0.002
6&7 vs 5&8	AO vs AI	0.0003
2&3 vs 1&4	AO vs CI	0.007
2&3 vs 1&4	AO vs CO	0.084
2&3 vs 1&4	AO vs AI	0.107
6&7 vs 2&3	AO vs CI	<0.001
6&7 vs 2&3	AO vs CO	<0.001
6&7 vs 2&3	AO vs AI	<0.001
6&7 vs 1&4	AO vs CI	<0.001
6&7 vs 1&4	AO vs CO	<0.001
6&7 vs 1&4	AO vs AI	<0.001
5&8 vs 2&3	AO vs CI	0.014
5&8 vs 2&3	AO vs CO	<0.001
5&8 vs 2&3	AO vs AI	<0.001
5&8 vs 1&4	AO vs CI	<0.001
5&8 vs 1&4	AO vs CO	<0.001
5&8 vs 1&4	AO vs AI	<0.001

Table 3.6. Chemical composition of grain mix and chopped hay at the Beef Research Unit by treatment.

	Grain mix[1]			Chopped	
Nutrient[2]	Inorganic	Organic	SEM	hay	SEM[3]
DM, %	88.3	88.0	0.33	90.9	0.35
CP, % of DM	24.5	22.6	1.07	12.6	0.37
ADF, % of DM	12.1	11.5	0.63	45	0.80
NDF, % of DM	21.4	19.6	0.50	59.8	0.90
TDN, % of DM	79	79.3	0.89	57	0.93
NE_m, Mcal/kg	0.88	0.89	0.01	0.50	0.01
NE_g, Mcal/kg	0.59	0.60	0.01	0.25	0.01
Ca, % of DM	1.76	1.51	0.14	0.82	0.06
P, % of DM	0.92	0.81	0.05	0.23	0.01
Mg, % of DM	0.295	0.27	0.01	0.21	0.01
K, % of DM	1.54	1.40	0.11	2.27	0.12
Na, % of DM	0.45	0.39	0.03	0.03	0.002
S, % of DM	0.32	0.29	0.02	0.14	0.01
Fe, ppm	421	381	40.69	148	20.3
Zn, ppm	179	173	14.05	21	0.81
Cu, ppm	34	29	2.51	8	0.70
Mn, ppm	144	140	6.75	28	1.11
Mb, ppm	1.05	0.73	0.18	0.35	0.12

Table 3.6 (cont.)

RFV			84	1.82
Co, added ppm	1	1		
Cu, added ppm	12	12		
Fe, added ppm	20	20		
Zn, added ppm	70	70		

[1]Grain mix: Inorganic trace minerals (Zn, Mn, Cu, Co) in sulfate forms. Organic trace minerals (Zn, Mn, Cu, Co) supplied by ZINPRO, MANPRO, Cu-PLEX, and COPRO.

[2]Feed samples were taken weekly from wk 13 to 20 and composited in two subsamples for each replicate.

[3]SEM= Standard error of the mean.

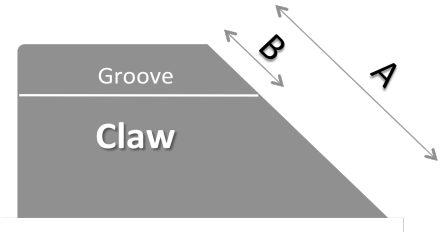

Figure 3.1. Depiction of how claw length (A) and groove length (B) were measured.

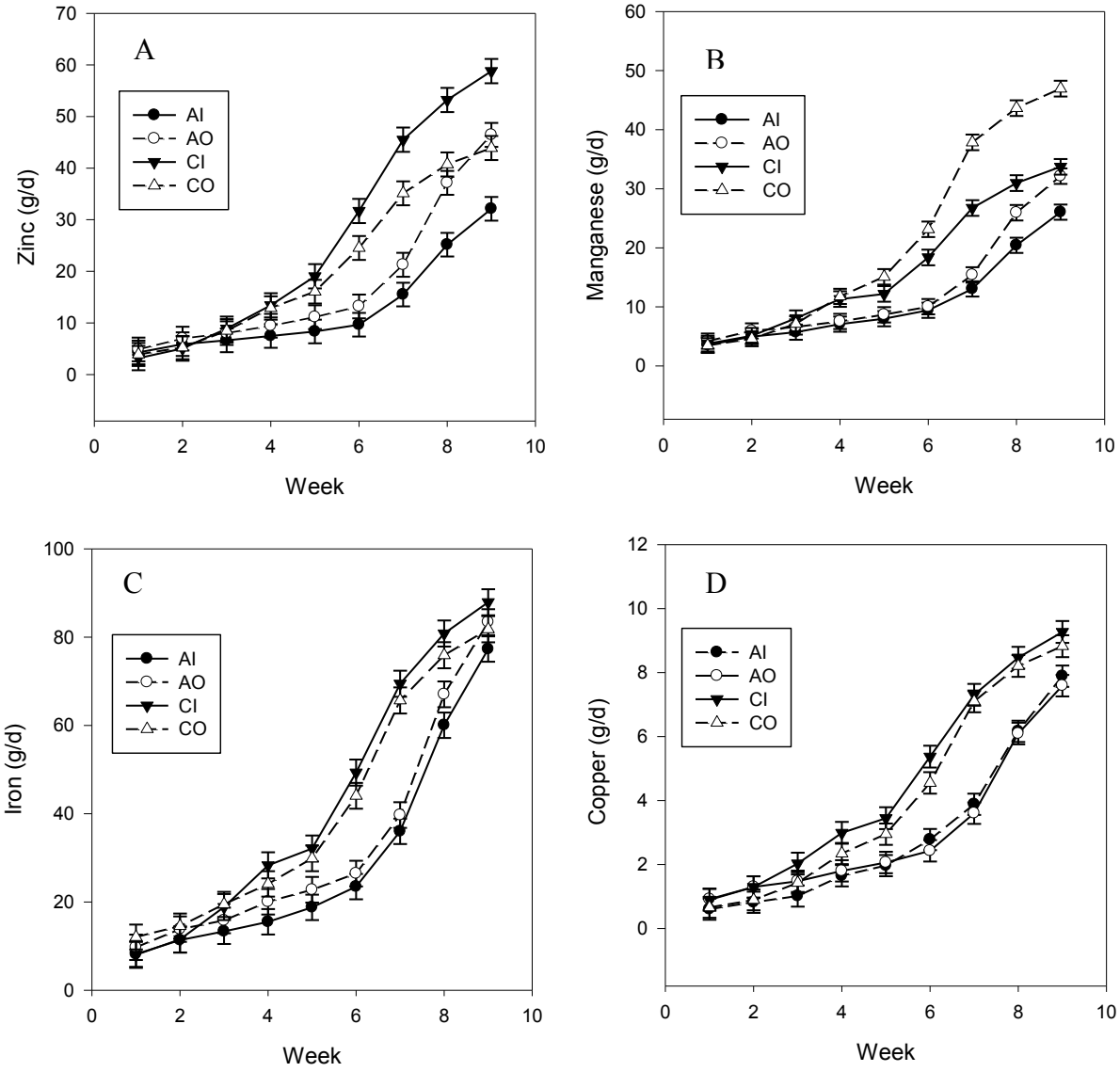

Figure 3.2. Mean daily intake of zinc (panel A), manganese (panel B), copper (panel C), iron (panel D), from wk 1 to wk 9 for calves fed conventional PN and inorganic TM (CI), conventional PN and organic TM (CO), accelerated PN and inorganic TM (AI), accelerated PN and organic TM (AO).

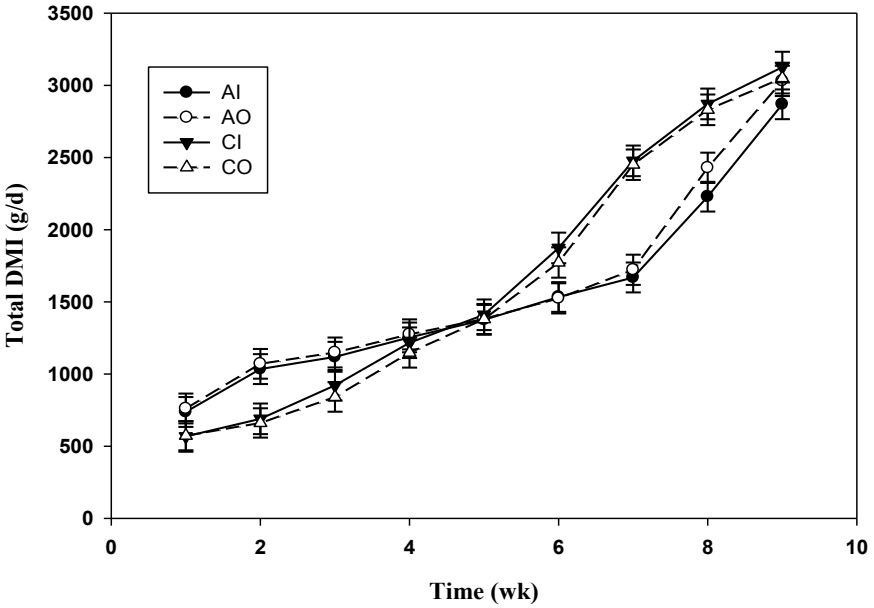

Figure 3.3. Mean total DMI from wk 1 to wk 9 for calves fed conventional PN and inorganic TM (CI), conventional PN and organic TM (CO), accelerated PN and inorganic TM (AI), accelerated PN and organic TM (AO).

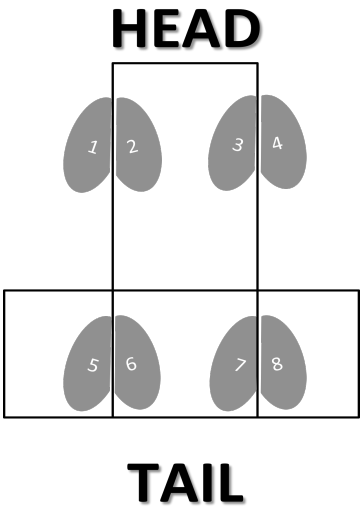

Figure 3.4. Initial claw length was greater for medial claws than for lateral, and for rear claws vs front claws.

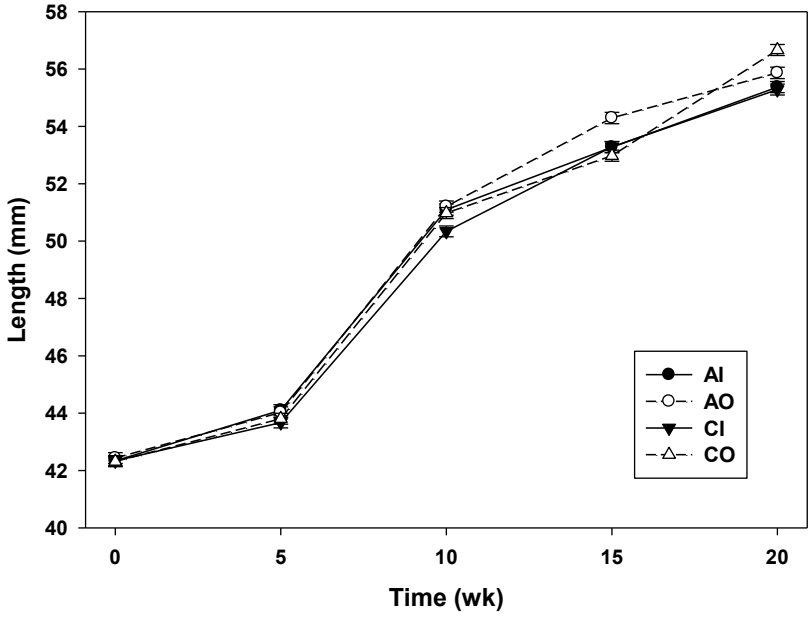

Figure 3.5. Mean claw length of calves fed accelerated inorganic (AI), accelerated organic (AO), conventional inorganic (CI), or conventional organic (CO) treatment combinations from 10 to 20 wk. The treatment effects PN (P=0.06) and TM (P=0.006) were significant.

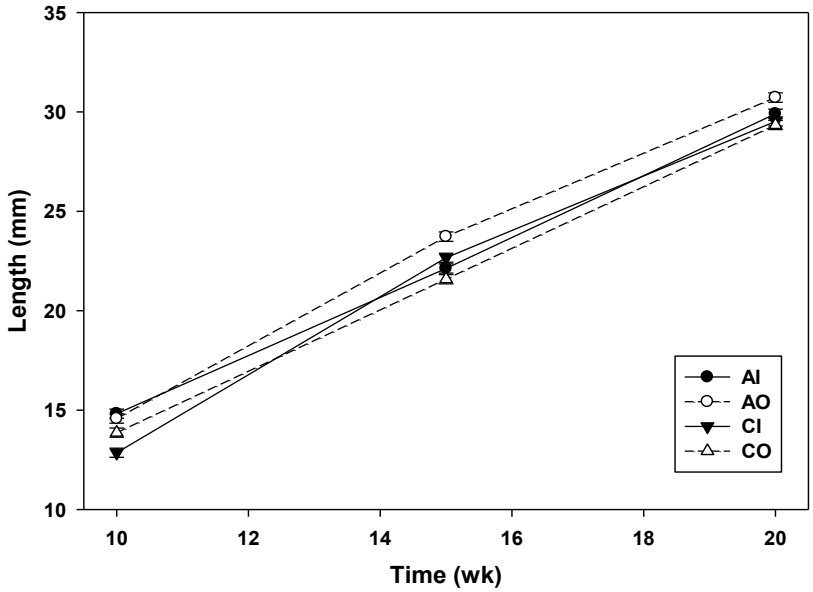

Figure 3.6. Mean groove length of calves fed accelerated inorganic (AI), accelerated organic (AO), conventional inorganic (CI), or conventional organic (CO) treatment combinations from 10 to 20 wk. The treatment effect PN (P<0.0001) and AO (P=0.008) were significant.

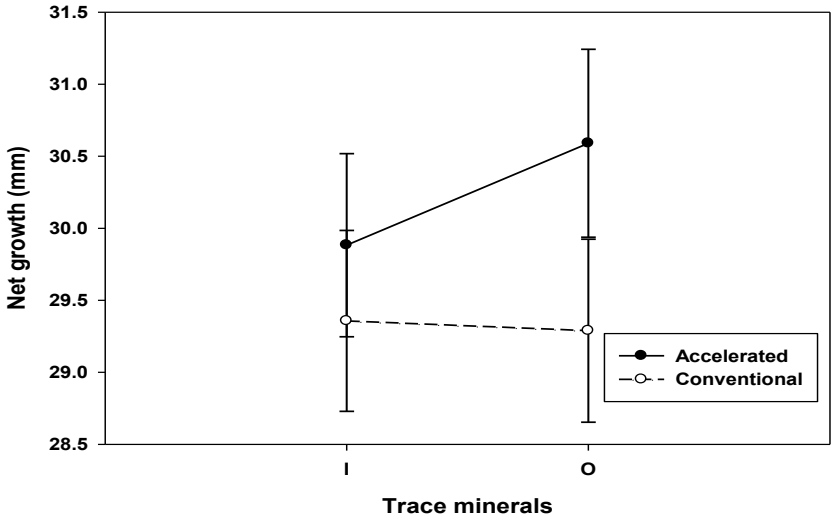

Figure 3.7. Mean growth interaction of PN and TM treatment effects. During the period from wk 0 to wk 20.

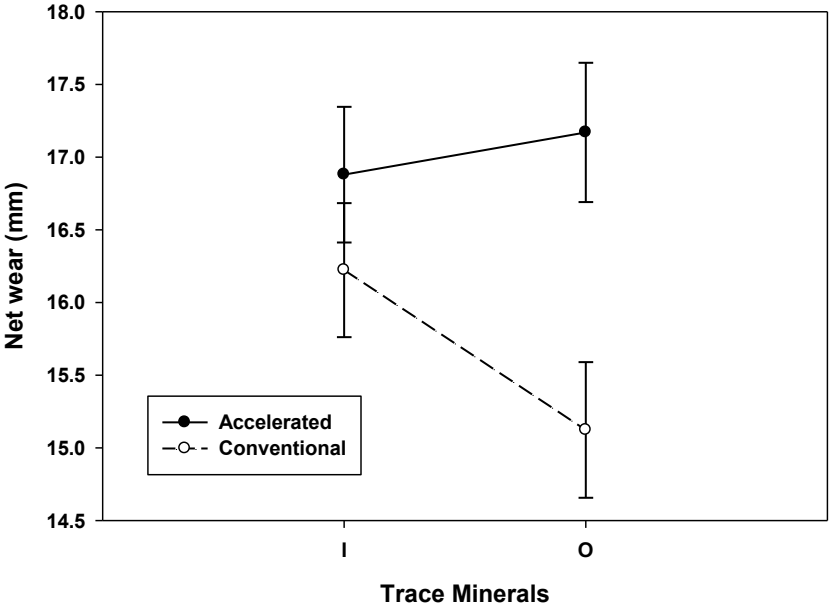

Figure 3.8. Mean net wear interaction of PN and TM treatment effects. During the period from 0 to 20 wk. The treatment effect PN was significant (P=0.005).

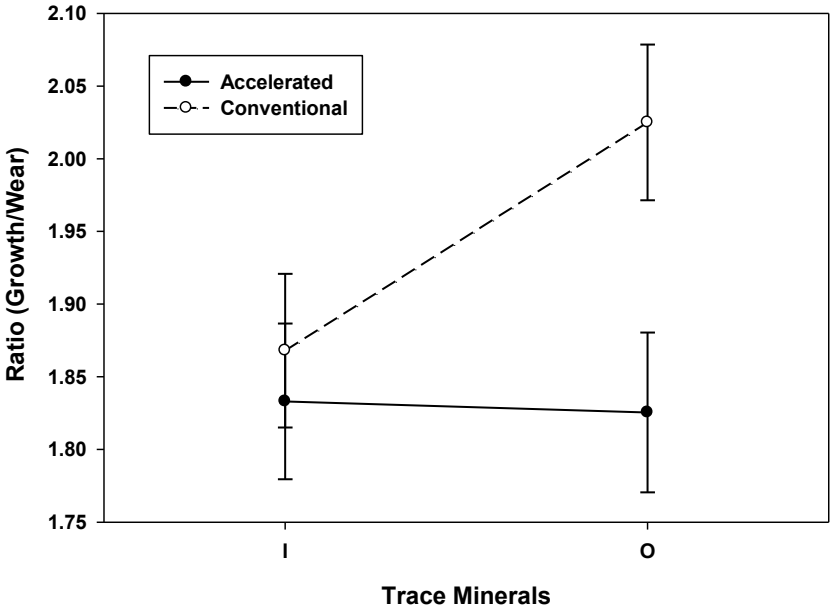

Figure 3.9. Mean ratio growth/wear interaction of PN and TM treatment effects. During the period from 0 to 20 wk. The treatment effect PN (P<0.05) and CO (P<0.05) were significant.

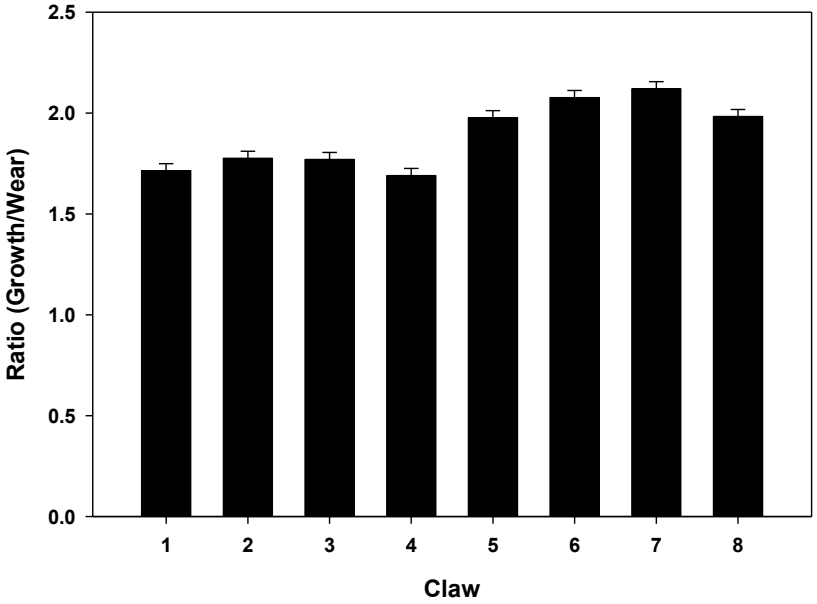

Figure 3.10. Ratio Growth/Wear by each claw in the period from 0 to 20 wk. Claw 6 and 7 greater (P=0.006) than others.

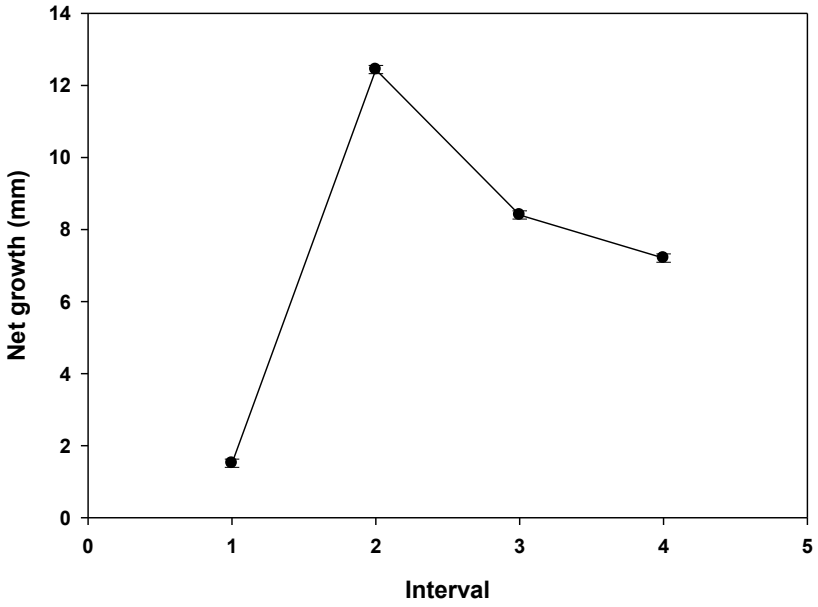

Figure 3.11. Mean claw growths by interval time for all calves. Interval 1 from 0 to 5 wk, interval 2 from 5 to 10 wk, interval 3 from 10 to 15 wk, interval 4 from 15 to 20 wk. The PN*TM*Interval was significant (P<0.0001).

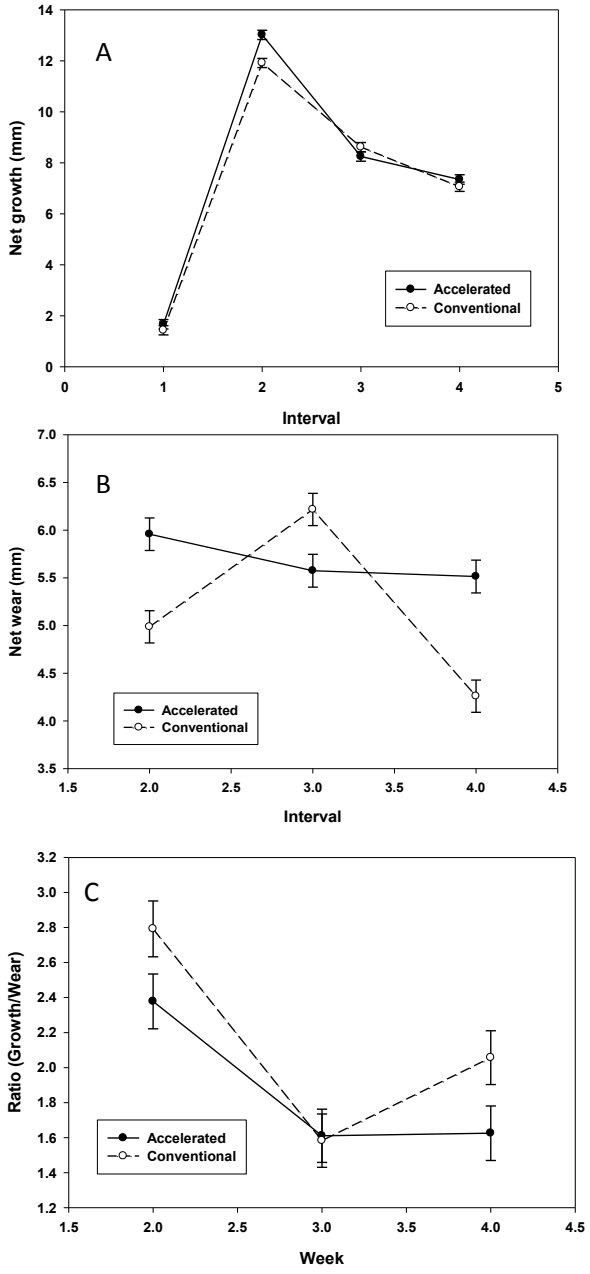

Figure 3.12. Mean growth by PN effect at each interval time (panel A), wear by PN effect at each interval time (panel B), and ratio growth/wear by PN effect at each interval time (panel C). Interval 1 from 0 to 5 wk, interval 2 from 5 to 10 wk, interval 3 from 10 to 15 wk, interval 4 from 15 to 20 wk.

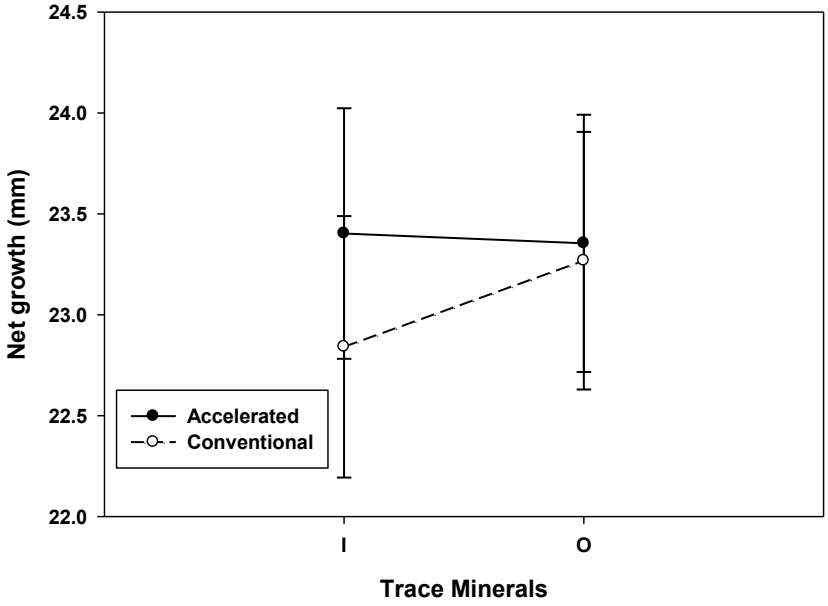

Figure 3.13. Mean residual net growth interaction of PN and TM at 35 wk.

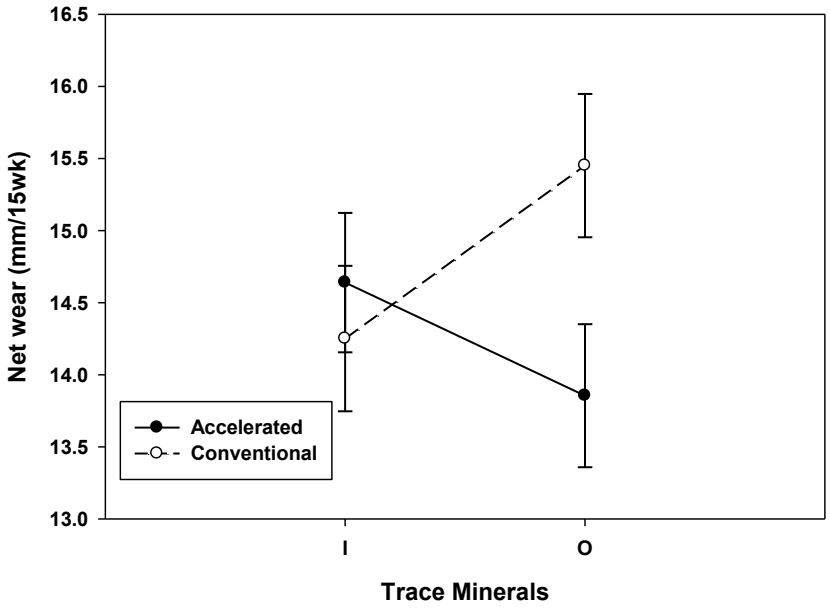

Figure 3.14. Mean residual net wear interaction of PN and TM at 35 wk

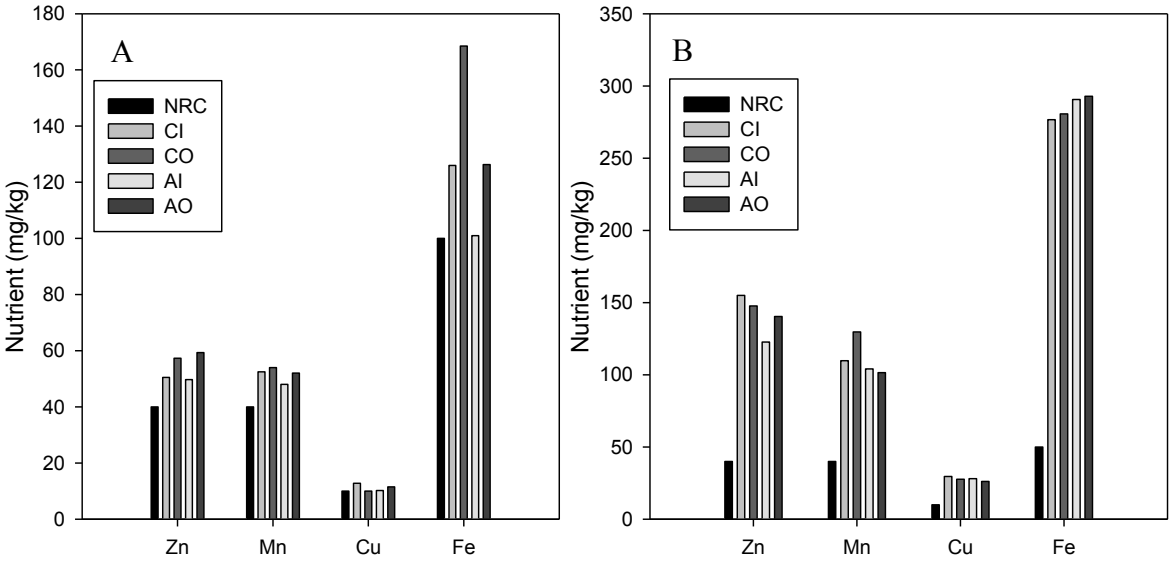

Figure 3.15. Trace mineral concentrations of Zn, Mn, Cu, and Fe in milk replacer (panel A) and starter (panel B) recommended by the NRC, and chemical analysis concentrations for feeds containing conventional PN and inorganic TM (CI), conventional PN and organic TM (CO), accelerated PN and inorganic TM (AI), and accelerated PN and organic TM (AO) treatment effects.